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Bioelectrical Phenomena in the Cortex
of the Larger (Cerebral) Hemispheres.

Part I.

by

A.I. Roitbak

Preface

The conditioned reflex is a central physiological phenomenon
in the normal work of the cortex of the larger hemispheres. (TN:
Hereafter referred to as the cerebral cortex.) Proceeding from this,
the main gap in the electrophysiological study of the cerebral cortex
can be formulated in approximately the following way: to study on
the basis of bioelectrical expressions of activity of cortical neurons
those inner nerve processes which are the basis of the conditioned-
reflex activity. (Footnote: Adrian thinks that... "the mechanism
of the conditioned reflex cannot be determined in terms of neuron,
synapse, and impulse' " (Adrian, 1938). The conditioned reflex is a

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reflex to be realized through temporary connection, i.e. this is a

physiological phenomenon that has definite structural foundations,

and certainly the mechanism of conditioned reflexes can be disclosed

by physiological methods of investigation (see Pavlov, 1932, 1934;

Beritashvili, 1953). On the other hand, in the opinion of Walter,

this is only a question of technical refinements in order to be able

to observe in the form of electrical discharges ideas that arise in

the brain of man (Walter, 1952). Of course, this notion is not

correct. Thinking cannot be expressed as adequately ideal in

bioelectrical potentials of brain tissue and cannot be reduced to them.)

Apparently the oscillographic method giving an opportunity for

direct observation of the nerve processes must play an incomparably

greater role than the method of extirpation and the method of

electrical stimulation, even in case it is limited by the use of

bioelectrical phenomena as such in the objectives of investigation of

the physiological functions of the cerebral cortex, the question of

the essentiality of the nature of these potentials, i.e. whether

temporarily it is not to be regarded as the physico-chemical bases

of these potentials, being put aside. Indicative of this are the

large-scale discoveries made during the last 25 years, after the

well-known paper of Samoilov (1930), in the field of the physiology

of the spinal cord, as well as the quick accumulation of facts on

the electrophysiology of the cerebral cortex.

Having set as my purpose the use of the oscillographic method for study of conditioned-reflex activity, I resolved to carry out a whole preliminary series of investigations in conditions of pointed, lingering experiments on narcotized and on normal animals and to study bioelectrical reactions of the cerebral cortex arising during its direct electrical stimulation and at stimulation of the receptors or the corresponding nerves. These reactions have been insufficiently studied even in conditions of ingenious experiments, and on normal animals they have not been able to be recorded until very recently. As to the origin and physiological importance of these reactions, there have been a number of hypotheses, often contradictory. It is necessary to think that only after solving these problems will it be possible to proceed to oscillographic investigation of the conditioned-reflex activity of the cerebral cortex.

The work presented is experimental, and little space is assigned to considerations not based directly on facts. No goal has been set to give a systematic literary survey of all that has been done in the sphere of the electrophysiology of the cerebral cortex. Special attention has been allotted to clarification of certain complex, debatable questions. Although each cycle of the investigations issued from the preceding and the separate parts of the work have logical

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connection with one another, it is difficult for them to appear
unified under a common designation. In Part I the results are
presented of pointed experiments on narcotized animals. In Part
II (not in this book) the results will be presented of experiments
on normal animals.

I consider it a pleasant duty to express profound thanks
to my instructor, Academy Member I.S. Beritashvili, for the
interest which he showed in my work and for his valuable instruction
and advice, and to Professors N.N. Dzidzishvili, A.B. Kogan, P.O.
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Chapter I

Certain Data From the Electrophysiology of the Nervous System

Which Will be Used During Analysis of the Bioelectrical Reactions

of the Cerebral Cortex

1. Regional Excitation and Local Potential

The School of Physiology, Leningrad University, contrary to
the prevalent principle of "all or nothing", has permitted various
modifications by which the state of excitation can be expressed
(see Ukhtomskii, 1939-40). According to the concepts developed in
this school, excitation does not obligatorily make off in the form

of a wave from the region of its origin. In certain conditions it
may keep to the place of origin for a more or less long time in the
form of a fixed, regional excitation, ready to flare up in the form
of an excitation wave (Ukhtomskii, 1927, 1932, 1939-40). Yet
Chagovets (1906) for purely theoretical reasons recognized the need
of a preliminary regional potential for the arising of spreading
excitation. Erlanger and Gasser in 1937 wrote that the electrotonic
potential is a certain unique electrical phenomenon which precedes
(at electrical irritation of a nerve) the current of spreading
excitation ("peak").

The electrotonic potential that arises at electrical irritation
of a nerve is connected, as supposed, with the capacitative properties
of the fibers (Erlanger and Gasser, 1937; Hodgkin, 1938). The
electrical potential quickly (after 50 microseconds) reaches a
maximum and is exponentially extinguished. To its characteristics
should be added that it grows in proportion to the intensity of the
stimulating current, that at reversal of the terminals of the
stimulating current it changes its sign, that temperature changes
hardly affect it, and finally that it radiates with logarithmic
decrement along the nerve fibers.

The local potential of the nerve fiber was recorded in 1938
by Hodgkin. He was unable to discover the local bioelectrical
potential in the nerve as a whole because of the very strong

polarization developed by all fibers and masking the local responses
that arise in certain fibers at subthreshold stimulation. He
succeeded at this for isolated nerve fiber of crab. This fiber,
which had a diameter of 25 microns, was deprived of the myelin sheath
that also had a positive side, since the polarization potential is
expressed more highly and radiates considerably further in medullated
fibers. The fiber was stretched with two pairs of forceps and placed
on three electrodes: one stimulating and two deflecting. At gradual
intensification of the irritating stimuli (the cathode on the fiber)
the following phenomena were observed.

At very weak stimulations only polarization arose. When the
energy of the stimulation equaled approximately 0.5 of the threshold
(for provocation of spreading excitation), then the potential
changed its character: the regular exponential curve was complicated
by supplementary fluctuation; the potential being recorded was a
combination of catelectrotonus and local potential. At subtraction
of the polarization potential from this total potential, it is possible
to determine the character of the local potential: it quickly increases
(for a period of 0.27 milliseconds), then gradually dies, lasting about
1.0 millisecond (Fig. 1, textpage 6: Local potential of nerve fiber.
Bioelectrical potentials are recorded that arise in the nerve fiber
(of crab) around the irritating electrode; stimulating is an electrical

impulse of short duration. Recordings A and B with irritability

energy of 1.5 conditional units: A is the stimulating cathode

electrode; B is the stimulating anode electrode. Recordings C and

D are with an energy of 1.0; E and F are with an energy of 1.0 over

a certain time, with reduction of the excitability: E is the

irritating cathode electrode, F is the anode. G and H are with an

energy of irritability of 0.61.

In the lower illustration recordings E and F are presented in

enlarged form; the cathode polarization potential is indicated by

dotted line. The a is the curve of the local potential, obtained

after deduction of the polarization potential from the total effect

at the cathode (Hogkin, 1938).).

At intensification of irritation the local potential was

increased and became somewhat more prolonged, i.e. the amplitude of

the local potential is graduated in connection with the change of

energy of stimulation.

When the amplitude of the local potential reached 15-20

millivolts (i.e. 0.3 of the current amplitude of the spreading

excitation), then it overincreased into an excitation current

that was expressed in the arising of a two-phase potential (40-60

millivolts): the excitation spread along the fiber and, passing

under the first deflecting electrode, reached the second (Fig. 1).

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The peak at first has the same course as the local potential:
the local potential overincreases into a current of spreading excitation
usually when it reaches its summit, i.e. the peak arises approximately
0.3 millisecond after the moment of stimulation. Thus, the latent
period of the excitation current (spreading) is determined by the time
that is necessary for the local potential to reach its maximum. As
for the latent period of the local potential in response to the
electrical stimulation, it is extremely small and equals 50-80 micro-
seconds (0.05-0.08 millisecond).

The local potential spreads along the fiber for a distance
of several millimeters. It spreads further than the polarization
potential. Hodgkin assumes that the mechanism of local-response
spread (regional excitation) differs substantially from the spread
of the polarization potential (the electrotonus).

Katz was able to detect local potential in the fibers of whole
sciatic nerve of frog; its duration proved equal to 0.5-0.6 millisecond
(Katz, 1947).

Castillo and Stark (1952) on isolated motor-nerve fiber of the
sciatic nerve of frog recorded a local potential at subthreshold
electrical stimulations of the fiber. The period of the local potential
of the medullated fiber proved equal to 0.4-0.7 millisecond; spreading
excitation arose at attainment by the local potential of an amplitude

equal to 1/4 of the amplitude of the effective current. Excitability

fluctuations ("spontaneous") were far greater than in the demyelinated

fiber. According to their data, the local potential at electrical

stimulation of isolated fiber arises in the region of Ranvier's node

and is recorded just from Ranvier's node and not from the internodal

section.

Finally, local potentials were detected in myelinated fibers

of spinal-cord roots of cat (Rosenblueth and Ramos, 1951). At

repeated subthreshold stimulations with the intervals between the

stimulations equal to 0.1-0.2 millisecond the phenomena were detected

of the summation of local potentials; during certain conditions, with

a certain intensity of stimulation, and after a certain number of

repeated stimulations, the effective emission current arose.

On the basis of a study of the changes of excitability at

different points of the nerve after application to the nerve of a

subthreshold shock of stimulation it was concluded that in nerve

fibers of frog the regional process of excitation spreads with

decrement to a distance of up to 9 mm. from the place of stimulation

(Karaev, 1938; Fudel'-Osipova, 1953).

According to the data of Fudel'-Osipova (1953), the increased

excitability in the nerve of frog after application of subthreshold

shock of stimulation lasts 1.5-4 milliseconds. Thus, from comparison

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of the data of Castillo and Stark and of Fudel'-Osipova, it can be
concluded that increased excitability after subthreshold shock of
stimulation lasts longer than the local potential. After the phase
of increased excitability there is observed a short-term phase
(1.5-2.5 milliseconds) of reduced excitability; in addition, 8-10
milliseconds after return of the nerve to the initial functional
state a new period of increased excitation is observed. This
period of increased excitability lasts 4-6 milliseconds, i.e. it
is more prolonged than the first, but, in addition to that, the
degree of excitability increase is considerably less. It should
be noted that the sequence changes of excitability in the nerve
that set in after the spreading excitation have, as known, a definite
electrical expression in the form of sequence potentials (Vorontsov,
1926; Erlanger and Gasser, 1937); it is still impossible at present
to link the sequence fluctuations of excitability after regional
excitation of a nerve, likewise detected by Fudel'-Osipova, with
certain bioelectrical phenomena, since after the local potential
in the nerve fiber no sequence potentials were recorded.

Several hours after the operation of the fiber removal and
after lengthy experimentation with it, it loses the capacity to
give spreading excitation - only a local potential arises at the
time of any energies of stimulation, then the stimulation causes
only the polarization potential (Castillo and Stark, 1952).

If the excitation spreading along a nerve reaches the parabiotic
part, it provokes in it regional excitation and the local potential
suitable to it; direct electrical stimulation of the parabiotic part
also causes regional graduated excitation (Vorontsov, 1952).

2. Forms of Bioelectrical Potentials in the Central Nervous System

Slow potentials. To slow fluctuations of the bioelectrical
potential belong fluctuations of duration over 10 milliseconds.

For up to 40 years the slow fluctuations of the bioelectrical
potential in the central nervous system has been explained usually
on the basis of certain electrical phenomena of the activity of the
axon. For instance, the components of the complex, long-term bio-
electric reaction arising in the optic tract of the cortex at
stimulation of the optic nerve and the positive or negative sign of the
separate components have been treated from the point of view of the
arrival of the impulses along the axons in the deflecting part and
the departure of the impulses along the axons from the area in question;
the bioelectrical potential being recorded has been considered the
resultant effective current of the mass of axon elements oriented in
various directions that have at each given point of the cortex a very
different source of origin (O'Leary and Bishop, 1938). Attempts to
analyze the slow biopotentials, deeming that they consisted of axonic
action currents emitted from the region of their arising and subordinated

to the principle of "all or nothing", have clashed with insurmountable difficulties.

At the present time it dan be considered demonstrated that the slow biopotentials do not consist of quick axonic action currents but represent a special type of activity (Renshaw, Forbes and Morison, 1940; Li Choh-Luh and Jasper, 1953) and that slow fluctuations of the biopotential in the central nervous system are the sum-total expression of elementary local potentials. These elementary local potentials arise in the bodies and dendrites of the nerve cells in the region of the synapses under the action of the impulses of excitation of the synaptic terminals (Beritov, 1945, 1949; Bremer, 1949).

The local potential expresses a regional, local excitation that arises in the neuron element under a synapse. (Footnote: Eccles uses the term "synaptic potential". However, it should be recognized, indeed as Beritov, that once this potential does not express the excitation of the synapse and expresses regional excitation of the cell, then it is unfitting to designate it a synaptic potential. There has remained for these potentials the designation "local potential", which will be used throughout the present work.) Regional excitation and the local potential corresponding to it are characteristic only to central nerve elements as spreading excitation and the current of action (peak) corresponding to it for the fibers of the peripheral

nerve, in which, as we have seen, the regional excitation arises
only during certain conditions of experimental reaction.

As was said, the local potential in the medullated fibers
lasts 0.5-0.7 millisecond and in the demyelinated 1-2 milliseconds.
In the neurons of the spinal cord the local potentials have a
considerably longer duration. From the gray matter of the spinal
cord of cat in the region of the posterior horn (segment lambda$_7$)
by needle electrode there were deflected, in response to threshold
stimulation of the tibial nerve, biopotentials the shortest duration
of which equaled 12 milliseconds (Beritov, Bakuradze and Roitbak,
1948). From the anterior horn of the spinal cord of cat at stimula-
tion of the corresponding motor nerve biopotentials were deflected
with a duration of 10 milliseconds (Brooks and Eccles, 1948). The
greater length of the slow potentials, recorded from the posterior
horn, is probably explained not by the fact that in the cells of
the posterior horns the local potential lasts longer than in the
motor neurons, but by the fact that at stimulation of the tibial
nerve a less synchronous discharge of impulses proceeds to the cells
of the posterior horn than to the motor neurons in case of stimula-
tion of the motor nerve containing fibers similar in conduction rate.
This probably stipulated a certain tentative summation of local
potentials in the cells of the posterior horns, which was expressed
in the fact that slow potentials were recorded of somewhat greater
duration. Moreover, Eccles used a more slender microelectrode, which

also should play a definite role in the result of the experiment.

Recently there has been success in effecting intracellular introduction of the discharge microelectrode into the motor neurons of the spinal cord (Brock, Coombs, and Eccles, 1952). In response to stimulation of the corresponding motor nerve from the motor neuron local potentials were discharged with a duration of 10 milliseconds.

Quick potentials. At the carrying off of biopotentials from the central nerve formations quick potentials are also recorded. The length of their sequence is from 0.5-1.0 millisecond, but, when they flow together, more prolonged fluctuations can arise, the complex character of which is discovered at quick survey. Quick potentials arise at excitation of the afferent fibers, their collaterals, and evidently the synaptic terminals during excitation of the axons of the intermediate and efferent neurons. They also arise during the discharge of the cells themselves (Eccles, 1953; Li and Jasper, 1953). The frequency of the quick potentials, i.e. of the impulses of excitation in the axon of the pyramidal cell of the cortex, can during strychnine poisoning reach 500-900 per second (Adrian and Moruzzi, 1939).

Electrical potentials arising in the central nervous system during regional excitation and during spreading excitation of its

neuronic elements are not epiphenomena devoid of physiological signifi-

cance. With regard to the current of action attending the spreading

excitation, this does not require special explanations if it is assumed

that the excitation spread along the nerve fibers proceeds by means

of the current of excitation and that the transmission of the excitation

from one neuron to another neuron likewise proceeds by means of the

excitation currents of the synaptic formations (see Ukhtomskii, 1939-

40; Beritov, 1948; Eccles, 1946, et al.). (Footnote: For the question

of the mechanism of transmission of excitation, just now not much can

be added to what Mislavskii wrote more than 50 years ago: "Finally,

it is difficult to deny the possibility of the development of any

chemical irritant at the point of contact of the terminal nerve

apparatus with the matter subject to excitation, but at not having

any factual data it is also difficult, if not even more difficult,

to prove it. The hypothesis of electrical action or discharge has

for itself a more tangible backing" (Mislavskii, 1895).)

To local potentials is now ascribed an extraordinary role in

the mechanism of the activity of the neuronic elements. Of course,

if the very important physiological importance of the biocurrent that

arises during spreading excitation is recognized, then already a

priori it is necessary to recognize the physiological importance of

the biocurrent arising during regional excitation.

At observation of the bioelectrical phenomena in the central
nervous system we run into two phenomena stipulated by the local
potentials in the neuronic elements.

When a local potential arises in the cells, then an electrotonic
reaction arises, first in those fibers which are axons of the activated
neurons and secondly in those fibers which terminate with synapses at
these neurons. The electrotonus spreads along the fibers with decrement
and during certain conditions can be detected (from the roots of the
spinal cord) at a distance of up to 10 mm. in the form of a negative
bioelectrical potential. Thus, the local potentials of the nerve cells
can stipulate the phenomenon of the physiological electrotonus of the
nerve elements.

However, there were also observed electrical phenomena of another
character that did not attract to themselves special attention, namely
at the arising of regional excitation in the cell from the axon a
positive potential was registered; for example, at stimulation of the
optic nerve a negative potential was discharged from the electrode found
in the outer geniculate body at the level of the layer of cellular
bodies; a positive potential at this time was discharged from the
electrode found at the level of the axons of these cells (Bishop and
O'Leary, 1943). Perhaps these electrical phenomena which we still
run into are phenomena essentially like secondary electrotonic changes,

the peri-electrons (sic! -should be peri-electrotoni?) (Vvedenskii,
1920), (Footnote: According to the data of Beritov and Roitbak (1954),
in the electrotonic and peri-electrotonic spheres of the nerve trunk
potentials of opposite sign arise. For example, at completion of a
descending current at the cathode a negative potential is registered
that gradually weakens at removing the deflecting electrode from the
cathode. Finally, at a certain distance it stops being registered.
If too the electrode is removed still further, then at completion
of the current a positive potential of considerable amplitude is
registered, and this sphere of the peri-electrotonus extends for a
considerable distance.) If this is so, then it is possible to make
the following conclusion: local potentials arising during regional
excitation of nerve cells can stipulate electrotonic and peri-
electrotonic phenomena in neuronic elements.

Is there the possibility of referring certain components of
the biopotentials to certain neuronic elements?

The first connection between the data of oscillography and the
data of morphology was established when it was successfully discovered,
on the basis of oscillographic analysis, that nerve trunks of different
diameter produce at excitation biocurrents of different length and
that the spread rate of these biocurrents (i.e. the excitations) is
different for fibers of different size. The quick-conduction fibers

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proved to be fibers of larger diameter (the A-fiber), the slow-conduction fibers were the thin C-fibers (Erlanger and Gasser, 1937).

The establishment of a second such connection was possible as a result of investigations of the bioelectrical potentials of the spinal cord.

3. Some Data of the Electrophysiology of the Spinal Cord

Cajal discovered that the posterior-root fibers are connected with the motor neurons of the corresponding side, first immediately through direct collaterals and secondly through intermediate neurons (Cajal, 1893a). On this basis Bekhterev concluded that, correspondingly, the spinal-cord reflexes can be accomplished in a twofold way: the nerve impulse can be transmitted directly to the motor cell of the anterior horn or the nerve impulse be transmitted to the intermediate neuron, which in its turn directly or by means of another intermediate neuron transmits the impulse to the motor neuron (Bekhterev, 1898).

At the beginning of this century it was found that coordination of the reflexes is realized in the posterior half of the spinal cord, which is very complexly organized, and that the cells of the posterior horns in a number of properties, for instance in sensitivity to strychnine, are distinguished from cells of the anterior horn (Beritov, 1910; see also Pavlov, 1912-13). The fine morphology of cells of the anterior horn and of cells of the posterior horn is different: the cells differ in size and shape, as well as in distribution of synaptic terminals on them (Cholokashvili, 1953).

As a result of oscillographic investigations on cats, it was found that direct effect on the motor neurons is realized through the thickest (12-20 microns) and that the quick-conduction fibers connected with the proprioceptors of the muscles, i.e. direct posterior-root collaterals leading to the motor neurons, were shown to have their origin from the proprioceptive fibers. The intermediate neurons engage in the action under the influence of impulses from fibers connected with the cutaneous receptors (Lloyd, 1943a, 1943b). (Footnote: Razdol'skii in 1924, on the basis of a comparison of the physiological characteristics of the tendon and cutaneous reflexes from neurological data, came to the conclusion "that tendon reflexes are realized by two-neuron reflex arcs, and that cutaneous (reflexes are realized) by the triple cutaneous and the polyneural".)

During the carrying off of biopotentials from the anterior and posterior roots of the spinal cord gray-matter potentials are disclosed (Mislavskii, 1894), which are electrotonically carried out along the root fibers (Barron and Matthews, 1938). For observation of these biopotentials it was most beneficial to place the root on the discharge electrodes so that one discharge electrode ("active") was in the cerebrum itself, but did not affect it, and the second was as far as possible from the brain. After Barron and Matthews this method was used by Bonnet and Bremer (1938-1950), Beritov and Roitbak (1947-1950), Vorontsov (1949, 1951), Eccles (1946), Roitbak (1950), Fuortes (1951), Kostiuk (1955), and others.

From the anterior roots, i.e. from the axons of the motor

neurons slow electrotonic potentials are registered that have been

stipulated by the local potentials of the motor neurons. Apparently

the electrotonic reaction of each axon is the consequence and

expression of the local potential, namely of the cell from which

it took its beginning.

When the motor neurons are activated only by excitation

impulses from the direct posterior-root collaterals (for instance,

at stimulation of the muscle nerve or at threshold stimulation of

a mixed nerve or of a posterior root), then the following electrical

effect is registered from the anterior root: a certain time after

the stimulation artefact a quick potential arises, after which a

negative slow potential follows. The initial quick potential is

the consequence of a relatively synchronous discharge of afferent

impulses and expresses the excitation currents of the presynaptic

fibers and of the synaptic terminals of direct posterior-root

collaterals in the anterior horn, being electrotonically carried

along the anterior-root fibers (Beritov, 1946, 1949). The negative

slow potential expresses local potentials in motoneurons, arising

below the synapses of the direct posterior-root collaterals.

A large number of synapses are on each motoneuron of the

anterior horn. After 100 of them are counted on the body of the

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motoneuron of the spinal cord of cat (Cholokashvili, 1953). Their
average size is 1 micron (Haggar and Barr, 1950). Apparently not
devoid of physiological importance is the fact that synapses are
arranged on the surface of the motoneuron in the form of separate
foci that are in their nature "the synaptic fields" of the cell
(Zurabashvili, 1947).

Excitation of one synapse is insufficient to arouse the cell
and to be discharged to its axon. It is likewise assumed that
insufficient for this is excitation of several synapses that remain
far from one another (Lorente de Nó, 1938). Excitation of a
motoneuron and discharge to the axon proceed when afferent impulses
come simultaneously to a whole group of synapses arranged on the
body of the cell in a certain proximity to one another. Otherwise,
only regional excitation arises in the cell, with a local potential
corresponding to it, as occurs in a nerve fiber at subthreshold
stimulation (Fig. 2, textpage 13: Local potentials of motoneurons
of the spinal cord of cat. A: A microelectrode is introduced into
the anterior horn in the region of a group of motoneurons of the
quadriceps muscle. A slow negative potential, 100 microvolts, 10
milliseconds (Brooks and Eccles, 1948), is registered in response to
a shock of stimulation applied to the quadriceps nerve. B: Potentials
from the 8th anterior root in response to a shock of stimulation to

the 8th posterior root. The upper curve is recorded on non-

narcotized animal. The second and third curves are recorded after

giving nembutal, 70 and 90 mg. (per kg. of weight), respectively.

C (TN: B looks like a beta in Russian; the 3d letter of the alphabet,

which I here call C, looks like a capital B): The same carrying

off; non-narcotized animal; the gastrocnemius nerve is stimulated.

The upper curve is the discharge of motoneurons, which arose 0.2

millisec. from the beginning of the development of the local potential.

The lower curve is the effect of the same stimulation, but at the

time of a state of inhibition of the motoneurons caused by the

preceding stimulation: only a local potential arises (Eccles, 1946).

For all illustrations stimulation artefact proceeds at first, then

fluctuation stipulated by arrival of afferent impulses proceeds at

first; after this fluctuation the local potential arises (in pure

form or complicated by the excitation current of the motoneurons).)

Thus, the arising of the discharge of the neuron assumes seizure by

the excitation of a certain territory of the cell body, as for the

arising of spreading excitation in a nerve fiber, seizure by regional

excitation of a certain length of the fiber is required (Rushton,

1937; Makarov, 1947).

In experiments with intracellular discharge of potentials of

the motoneurons it was ascertained that the discharge of the motoneuron

proceeds at attainment of a critical amplitude of 10-12 millivolts (1/10 of the amplitude of the peak potential of the motoneuron) by the local potential. It was concluded that at excitation of a synaptic terminal a local potential arises 1 millivolt in magnitude; thus, for a discharge of the motoneuron to have set in, simultaneous stimulation of a minimum of 10 synaptic terminals is required (Eccles, 1953).

A negative slow potential begins without an appreciable latent period after the initial quick fluctuation, usually still in the descending limb of the latter (for cat it begins 0.7 millisecond after the moment of the arising of a quick fluctuation). Hence, it is possible to conclude that the local potential of the cell arises under the action of a biocurrent of the synaptic terminals with a negligible latent period, as in the nerve fiber in response to its direct electrical stimulation, i.e. with the latent period measured by fractions of a millisecond. This fact must serve as one of the arguments in favor of the electrical theory of the transmission of an excitation from neuron to neuron.

The discharge of the motoneuron proceeds at the attainment of a certain critical magnitude by the local potential, i.e. by regional' excitation. The delay in the conduction of the excitation is stipulated by this circumstance and is determined by the time (2 milliseconds and more) which passes while the local potential that has arisen reaches

this critical magnitude (Eccles, 1946). Thus, there is essentially

the same phenomenon as in the nerve fiber, but different time relation-

ships.

    As has been said, duration of the local potential in the cells

of the spinal cord of cat equals 10 milliseconds. A biopotential of

greater length, of the order of 15 milliseconds (the rising phase

lasts 2.5 milliseconds), is discharged from the anterior root. This

is explained by the fact that at electrotonic "spread" of the cellular

potentials the duration of the potential is increased, its form is

somewhat distorted (Eccles, 1946), and its magnitude is sharply reduced.

    When the motoneurons are stimulated, this is expressed

oscillographically in this, that the slow potential is broken and a

quick biocurrent of great amplitude arises (Fig. 2). The slow potential

at the time of the discharge of the motoneurons can only weaken. This

needs to be understood as an expression of the fact that excitation

of only part of the activated motoneurons has proceeded. In those

which were not stimulated, local potentials continue to develop and

then to die out. However, excitation of the motor axons can proceed

even without the corresponding cells having been discharged: the

fibers can be excited under the stimulating influence of currents

stipulated by local cellular potentials. In this case the excitation

impulses of the motor axons arise and proceed on the background of a

slow potential (Beritov and Roitbak, 1947b). The anterior-root

axonic excitation currents in this case too arise at attainment

of a certain magnitude by the slow potentials and disappear, in

connection with prolonged stimulation, when the slow potentials

weaken to a certain magnitude. Thus, the setting in of the anterior-

root excitation currents depends strictly on the size of the slow

potentials (Beritov and Roitbak, 1950).

     In the anterior roots of the cervical portion, from which

the diaphragm nerve proceeds, in connection with each respiratory

cycle there arise a slow negative potential (electrotonic reaction)

and a group of quick (potentials). The latter without decrement

spread along the fibers of the diaphragm nerve. The slow negative

potential arises according to the phase of inspiration and diminishes

at the time of the expiration phase. Quick potentials arise mainly

on the ascending limb of the slow potential. They are lacking at

time of the expiration phase (Gesell, Hunter, and Lillie, 1949).

Thus, under the influence of impulsation (sic! -impulse excitation?)

from the respiratory center there arises in the motoneurons essentially

the same bioelectrical reaction as under the influence of the afferent

impulses. Intensified respiratory movements are associated with

intensified slow potentials and intensified discharges of impulses,

and, contrariwise, at weakening of the respiration owing to previous

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artificial hyperventilation, both components of the bioelectrical

reaction weaken. Thus, intensity of discharges arising periodically

in the diaphragm nerve under the influence of the "impulsation" of

the respiratory center proves dependent to a certain extent on the

intensity of the slow negative potentials discharged from the

corresponding anterior roots.

It was well known, even at the time of Sechenov, that if a

subthreshold stimulation of certain intensity, i.e. a stimulation

which does not cause reflex contraction of muscles, is applied to

a sensory nerve, then it remains without effect on the reflex center.

In experiments on cat, if within 10-15 milliseconds after the first

subthreshold shock of stimulation a second analogous stimulation is

applied, then it can cause a reflex (Creed and coworkers, 1932). The

curve of summation is identical, in form and in time relationships,

to the slow potentials discharged during these conditions of stimulation

from the anterior root (Lloyd, 1946). (Footnote: According to Eccles'

experiments (1946), the summation curve in regard to provocation of

the discharge of the motoneurons continues 10 milliseconds, i.e. it

continues for as long a time as the local potential lasts spontaneously

in the motoneuron (see also Kostiuk, 1953).) Consequently, at applica-

tion to one and the same sensory nerve of two excitation discharges

with the interval such that the second discharge of impulses reaches

the motoneurons in the period of the regional excitation beginning

in them, response to the second discharge is facilitated. Thus,

regional excitation of motoneurons has a relationship with the

phenomena of facilitation and summation. It has a direct relation-

ship with the mechanism of the arising of excitation impulses. As

in the nerve fiber too, local potentials here are "forerunners" of

spreading excitation current. Oscillographic investigations have

revealed the accuracy of the theoretical conditions, according to

which there arises in the nerve cells regional excitation, which

can be finely graduated according to incoming impulses and which

can flare up in the form of an excitation wave (Ukhtomskii, 1927,

1932, 1939-40). On the other hand, the concept of the regional

state of the central excitation, created by Sherrington and his

school, has received confirmation. As known, according to this

concept the central state of excitation arising in the motoneuron

under the influence of the discharge of the stimulating afferent

impulses grows over a period of several sigmas (TN: thousandths

of a second), reaches a maximum, and gradually weakens further,

lasting generally about 20 sigmas. The discharge of the motoneuron

proceeds then when the state of the central excitation reaches a

certain threshold magnitude; the so-called synaptic delay is the

time which is necessary for the central state of excitation to have

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reached this threshold magnitude (Creed and coworkers, 1932). As
we see, all phenomena which are found issuing from the hypothetical
idea of the central state of excitation are explained on the basis
of this which was clarified by direct study of the local potentials.

A long (about 100 milliseconds) positive sequence potential
is recorded after a slow anterior-root potential, even if it (the
latter) is not complicated by quick potentials or by supplementary
negative fluctuations. At the time of this sequence potential a
reduction is observed of the excitability of the corresponding
motoneurons (Brooks, Downman and Eccles, 1950). Thus, long resultant
positive potentials arise after regional excitation of neurons and
are associated with the reduction of their excitation.

At intensification of the stimulation of the nerve from the
anterior root double bioelectric effects are discharged (Fig. 3):
after the negative potential (or discharge) already considered, a
second slow negative potential (or discharge) follows. (Legend
to Fig. 3, textpage 17: The biopotentials of motor neurons of the
spinal cord of cat, which first arise under the influence of impulses
from the direct posterior-root collaterals and then under the
influence of impulses from the intermediate neurons. The potentials
are recorded of the anterior root of the spinal cord of decerebrated
cat, that arise in response to separate stimulations of the cutaneous

nerve of the corresponding side. A - weak stimulation. B - strong

stimulation (Brooks and Fuortes, 1952).) These data were confirmed

by experiments with intracellular discharge of potentials of the

motor neuron: a second negative fluctuation arose at a certain force

of stimulation of the sensory nerve. At intensification of the

stimulation the latent period of its arising was shortened (Eccles,

1953). The duration and amplitude of the second slow fluctuation

were very changed. It was established that the second fluctuation

was connected with the activity of the intermediate neurons and

expresses regional excitation of the motor neurons arising under

the effect of impulses of excitation from axons of intermediate

neurons. Impulses from intermediate neurons fail to be registered

from the anterior root because of their asynchronous admission.

This second negative fluctuation, which can be complicated by

especially powerful and frequent biocurrents of the anterior-root

fibers, is characterized by the following properties: 1) it arises

only during good functional state of the preparation; 2) it arises

in connection with intensification, as well as in connection with

repetitions of the stimuli; it grows at repeated stimulations; 3)

during prolonged stimulation this fluctuation primarily weakens,

i.e. this effect is subject to quick exhaustion[*]; 4) this fluctuation

and the discharges corresponding to it are extraordinarily intensified

under the effect of strychnine, whereas the first negative fluctuation

is not significantly intensified. All these peculiarities are

characteristic to the intermediate neurons (Beritov, 1948). (* The

quick exhaustion of the intermediate neurons as compared with the

motor perhaps is dependent on the fact that the first are distinguished

by their comparatively small size, i.e. they contain a relatively

small amount of protoplasm (Malon, 1932). There are facts indicative

that nerve elements which contain a larger amount of the system being

excited are exhausted later (Beritov, 1932).)

When the functional state of the spinal cord is poor and when

any intensity and frequency (up to 50 a second) of stimulation is used,

only short-term slow fluctuations are recorded, expressing regional

excitation that arises under the effect of impulses from direct

posterior-root collaterals (Beritov and Roitbak, 1947b; Eccles, 1946).

In the intermediate neurons at this time under the influence of the

afferent impulses there also arise only a regional excitation, local

potentials which can be detected at discharge of potentials from the

posterior roots.

At discharge of potentials from the posterior roots, two

incidents are distinguished: discharge from intact root and from

the central end of sectioned root. In the first case at stimulation

of the corresponding nerve at first a quick bioelectrical component

is registered, connected with the afferent impulses that proceed

along the root in question; after it follows a slow potential

connected with activation of the gray matter of the posterior half

of the cerebrum. The initial quick component of the bioelectrical

reaction was confirmed by a special study of Beritov and Roitbak

(1947a) and then by Lloyd (1949). It proved very complex in

character: to the biocurrents of the afferent impulses proceeding

under a pair of discharging electrodes is added a series of quick

biopotentials; it was found that the latter are carried out from

the cerebrum and discharge the excitation biocurrents of the

collaterals of the afferent fibers and of their synaptic terminals

(Beritov and Roitbak, 1947a).

It is known that the slow potential discharged from the

posterior root is an electrotonic reaction which arises as the

result of arrival in the spinal cord of a discharge of afferent

impulses, slow potentials identical in character being registered

both from the root along which these potentials reached the brain

and from adjacent roots. Since the work of Eccles and Malcolm

(1946) and Beritov and Roitbak (1947-1950) it has been possible

to consider most probable that the slow posterior-root potential

arises because of activation of the neurons at which the fibers of

the stimulated root terminate. (In the opinion of certain investigators

(Barron and Matthews, 1938; Lloyd, 1949; and Vorontsov, 1955) slow

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posterior-root potentials discharge the excitation of the presynaptic
fibers and/or synaptic terminals. For criticism of this idea see
Eccles (1950).) Activation of the neurons, the arising of regional
excitation (of local potentials) in them, stipulates electrotonic
reaction of the posterior-root fibers. When under the action of
afferent impulses in the body or in the dendrite of a given neuron
a local potential arises, this leads to the establishment of a variety
of potentials, to the arising of an electrical current, and to electrontonus
in those fibers which terminate with synapses at the neuron in question;
electrotonic reaction arises both in the fiber which was excited before
this and in that which was not in an active state. Thus, slow posterior-
root potentials are a composite expression of a great number of local
potentials arising in the intermediate neurons of the posterior half of
the brain, the slow potential, discharged from the root, expresses local
potentials namely of those elements at which the fibers of the root in
question terminate.

Thus, we can judge indirectly about the excitation of the
intermediate neurons and about the impulses proceeding from them,
through the anterior-root effects (supplementary negative fluctuation,
supplementary discharge). We can judge concerning regional excitation
of intermediate neurons directly on the basis of posterior-root slow
potentials.

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Through the works of Beritov and Roitbak (1948, Figs. 40-42)
it is known that, to begin with, from the anterior roots powerful
slow potentials can be discharged and at this time from the posterior
there are virtually no bioelectrical reactions. It is possible to
observe this at stimulation of the humeral nerve in frog. The axons
of the intermediate neurons of the humeral region of the spinal
cord, which are thereby being excited, terminate in the lumbar region
directly on elements of the anterior horns. In the second place,
from the posterior roots slow potentials can be registered of very
great amplitude and length, and at this time from the anterior roots
only weak, short-term potentials can be registered arising under
the effect of impulses from the direct posterior-root collaterals.
This is observed during strong stimulations of the sensory nerve when
the functional state of the preparation is poor. On the basis of
these facts, as well as on the basis of lack of parallelism in regard
to the intensity and temporal course of the potentials of the posterior
and anterior roots, it was concluded that the sources of their origin
were different. (Later Fuortes (1951) disclosed similar facts and
came to an analogous conclusion.) However, these facts indicate that
cerebral biocurrents do not spread diffusely along the spinal cord
and that within the brain electrotonic distribution too of biocurrents
along the fibers is possible only for relatively small distances.

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This latter conclusion requires explanations. That electrotonic distribution of the currents within the brain is possible for short distances demonstrates the fact of the arising of an electrotonic reaction in the anterior and posterior roots: well then, the anterior- and the posterior-root fibers proceed a certain distance within the brain from the cells to the exit from the brain (or, contrariwise, from the entry into the brain to the place of termination at the cells). The fact that the slow potentials discharged from the anterior root at stimulation of the humeral nerve are not registered from the corresponding posterior one shows that within the brain along the direct posterior-root collaterals (which terminate on the motor neurons) electrotonic spread of the potentials occurs with such decrement that they do not reach the place of entry of the posterior-root fibers into the brain. As said, powerful slow posterior-root potentials do not register from the anterior roots. This shows that the electrotonus cannot spread along the axons of the intermediate neurons because, otherwise, it would be detected in the anterior roots (as the afferent impulse is detected that arrives at the motor neurons through direct collaterals).

Apparently a biopotential arising in the nerve cell can be detected only at a slight distance from it. Supportive of this concept too is the fact that the amplitude of the local potential of the motor

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neuron at intracellular discharge equals 10 millivolts and at extra-

cellular discharge from the nucleus of motor neurons 100 microvolts

(Fig. 2,A), i.e. in a 100 times lesser magnitude.

4. Concerning Long Nonfluctuating Bioelectrical Potentials

As far back as in early investigations of the bioelectrical

phenomena of the central nervous system, carried out with the aid

of a galvanometer, similar potentials were reported. Mislavskii

(1894, 1900), at the discharging of current from the posterior

roots of the spinal cord of frog observed long nonfluctuating

biocurrents during tetanic irritation of the sciatic nerve and

at adequate stimulations of the skin.

Delov and Lapitskii recorded during discharge of currents

from the spinal cord (1 electrode on the surface of the lumbar part

of the spinal cord, 2 on a crosswise section) the following phenomena:

quick fluctuations following the rhythm of stimulation of the sciatic

nerve up to 100 per second, were placed on a background of a slow

nonfluctuating potential, the amplitude of which was increased with

increase of frequency of stimulation up to 100 a second and reached

1 millivolt (Delov and Lapitskii, 1935). In Fig. 4, D is presented

the oscillographic recording of a nonfluctuating biopotential,

registered from the posterior root during tetanic stimulation of the

sciatic nerve.

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(Legend to Fig. 4, textpage 20: Long nonfluctuating potentials generated by neurons of the posterior half of the spinal cord during tetanic stimulation of the sensory nerves. Curarized frog with spinal cord revealed and circulation undisturbed. 12°C. Potentials are discharged from the 9th posterior root at a distance of 1 mm. from the brain. Direct-current booster. Recording by string oscillograph. A - the trifacial nerve of the opposite side is stimulated; frequency of stimulation 10 per second. B - frequency of stimulation 100 per second. C - the sciatic nerve of the corresponding side is stimulated; frequency of stimulation 10 per second; the beginning and end of brief stimulation. D - frequency of stimulation 100 per second; beginning and end of brief stimulation. Time marks for 10 milliseconds. (Roitbak, 1950).)

During tetanic irritations of the sensory nerves or of the posterior roots, as well as during adequate stimulations, for instance, of the muscle receptors a long nonfluctuating potential is registered from the anterior roots (Barron and Matthews, 1938). In Fig. 5 are presented recordings of the bioelectrical reaction of the anterior root of strychninized preparation of frog in response to tetanic stimulation of the sciatic nerve. As seen, a nonfluctuating potential arises, on the background of which the fluctuations are arranged according to the rhythm of the stimulation. At cessation of stimulation

the potential gradually weakens, but even after 4 seconds does not

reach the abscissa. It is certainly sustained by "impulsation" from

the intermediate neurons. There is aftereffect too in regard to

quick fluctuations (Beritov, Kvavilashvili, and Roitbak, 1950).

An analogous recording was made by Fuortes (1951).

Thus, the central nerve elements at arrival of frequent

impulses of excitation to them generate long nonfluctuating potentials

that certainly reflect the nonfluctuating state of the regional

excitation.

(Legend to Fig. 5, textpage 21: Prolonged nonfluctuating

potential generated by motoneurons of the spinal cord. Spinal

strychninized preparation of frog. 10°C. Potentials are discharged

from the 9th anterior root at a distance of 3 mm. from the brain.

Constant-current booster. Recording by string oscillograph. The

sciatic nerve of the corresponding side is stimulated; frequency of

stimulation 40 per second. A - beginning of stimulation. B - end

of stimulation and aftereffect. C - length of aftereffect 1 second

after recording of B. Recording of D was made 3 seconds after C

(Beritov, Kvavilashvili and Roitbak, 1950).)

At stimulation of the branch of the trifacial nerve in frog

from the posterior roots of the spinal cord are recorded considerable

slow potentials. At a stimulation frequency of 10-100 per second

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the potential acquires a nonfluctuating character (Fig. 4, A and B).

Thus, excitation impulses come from the cerebrum to the spinal inter-

mediate neurons in the cord, because of which an electrotonic reaction

arises in those fibers of the posterior root that form a synapse with

these neurons. Stimulation of the trifacial nerve usually does not

cause considerable bioelectrical reaction of the anterior root even

after strychnine poisoning of the spinal cord when stimulation of

the peroneal nerve causes most intense convulsive anterior-root

discharges (Roitbak, 1950). It would be possible to think that

impulses proceeding along the descending courses at stimulation of

the trifacial nerve are subthreshold for the intermediate neurons,

for instance, because of the fact that the corresponding synapses

are placed at a greater distance from one another. But then summation

phenomena would be expected at a combination of stimulations of the

trigeminal nerve and of the peroneal nerve. On the contrary, it

appears: if the peroneal nerve is stimulated on a background of

tetanic stimulation of the trigeminal nerve, causing a nonfluctuating

posterior root potential, then the reflex from stimulation of the

peroneal nerve proves delayed. Thus, stimulation of the trifacial

nerve, causing a slow potential in the character of a single bio-

electrical reaction in the spinal cord, generated by the neurons of

the posterior half of the spinal cord, stipulates inhibition of the

reflex activity of the spinal cord (Roitbak, 1950a).

The concept of the causal connection of the central inhibition with the slow bioelectrical potentials has been expressed by a number of physiologists (see Beritov, 1948). However, it is interesting that it is possible to reach this conclusion on the basis of the data obtained by Sechenov (1882). Sechenov in his outstanding investigation of electrical phenomena in the medulla oblongata ascertained that tetanization of sensory nerves leads to inhibition of "spontaneous" discharges in the medulla oblongata. The following from the phenomena described by him deserves special attention. Tetanic stimulation of a nerve leads to "deviation of the magnet, so that it remains diverted to the negative side even during further tetanization", i.e. in the medulla oblongata a nonfluctuating potential arises and inhibition thereby occurs of the "spontaneous" discharges. In this work of Sechenov, devoted to the study of inhibition on the basis of galvanic phenomena, we first find indication of the connection between the inhibition and the nonfluctuating long bioelectrical potential, to which none of his commentators has turned his attention.

In experiments with intracellular discharge of biopotentials of motoneurons it was established that when inhibition of afferent impulses comes to a motoneuron, then a positive potential is registered from its body (Brock and coworkers, 1952; Eccles, 1952, 1953). (Footnote: See Roitbak, 1955, Kostiuk, 1955, and Motsnyi, 1955, on the question of the electrical phenomena of the inhibition process.)

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Thus, during analysis of the bioelectrical potentials of the
spinal cord it proved possible to explain their physiological
importance and to link certain components of these potentials with
the activity of certain morphological formations. For instance,
we saw 3 components of the anterior-root potentials successfully
referred to the activity of the posterior-root collaterals, motor
neurons, and intermediate neurons.

It is an incomparably more complex matter than the inter-
pretation of the bioelectrical potentials of the cerebral cortex
at an attempt to refer these or other components of the bioelectrical
reactions of the cortex to the activity of certain neuronic elements
of the cortex, of that portion of the central nervous system which
is most complex in organization. However, only by proceeding in such
a way can the origin and importance be explained of the bioelectrical
reactions of the cortex and, likewise, can the electrographic method
be used for study of the physiological processes and phenomena of
the cortex. On the other hand, at solving this problem it also
becomes possible to refer these very processes and phenomena to
certain morphological bases and then, to use Pavlov's expression,
the dynamic phenomena that break out in the cortex can be coordinated
to the very fine details of the construction of the apparatus (Pavlov,
1932). It is impossible not to agree with Pavlov in this, that
during a study of the cortical activity only those concepts which

are characterized as spatial concepts have likelihood of mastering

the subject (Pavlov, 1912, 1913).

Chapter II

Bioelectrical Potentials Arising in the Cerebral Cortex During

Direct Electrical Stimulation of its Surface

The cerebrum differs from the spinal cord by, among other things,

the fact (and this has drawn the attention already of the first investi-

gators of the electrical phenomena of the central nervous system) that

during the absence of special stimulations and during deliberate

exclusion of external stimulations from the cerebrum and from the

cerebral cortex, in particular, certain electrical fluctuations are

discharged. Evidently the so-called "spontaneous" electrical activity

of the cerebrum is a consequence and an expression of the greater

excitability of its nerve elements, in comparison with the spinal

neurons. Apparently various negligible external and internal stimula-

tions are capable of causing excitation of the neurons that compose

the nerve centers of the cerebrum. This should particularly be

referred to cortical neurons possessing highest excitability. We

shall come back to this question again.

As for the specially provoked bioelectrical reactions of the

cerebral cortex, this question too comprises the main content of the

present investigation. The following are experimental possibilities

for excitation of the cortical neurons by means of nerve impulses

the source of which can be determined.

a) Excitation of afferent systems of the cortex by adequate

stimulations of the receptors or by electrical stimulation of the

corresponding sensory nerves.

b) Excitation of the system of callosal fibers by electrical

stimulation of the cortex of the opposite hemisphere or of the corpus

callosum itself. It should be noted that Danilevskii was the first

to observe bioelectrical reactions in the cortex at stimulation of

the cortex of the opposite hemisphere (1891).

c) Excitation of the system of fibers in layer I of the cortex

by direct electrical stimulation of the surface of the cortex.

d) Finally, it is possible to send antidromically excitation

impulses into the pyramidal neurons of the cortex during stimulation

of the pyramidal tracts (Woolsey and Chang, 1947).

The first two possibilities were used during a study of electrical

phenomena in the cortex even in the last century and at the beginning

of this century. With the development of an oscillographic technique

quite a large number of similar investigations appeared (see Chapter IV).

In world literature until recently there were only 2 articles

relative to the bioelectrical reactions of the cerebral cortex that set

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in at the time of direct electrical stimulation of its surface. The

first such investigation was made by Adrian (1936). Some additional

facts were reported in Rosenblueth and Cannon's work (1942). In

1950 I presented a paper at a session of the Georgian Physiological

Society on a method I had worked out for the set-up of such experiments

and on the characteristics and source of cortical bioelectrical

potentials that arise at direct stimulation of the surface of the

cortex (Roitbak, 1950b). In 1951 Chang, having used a similar method

(which he described in detail and does not substantiate), published

a number of facts analogous to those on which I reported. Recently

articles have appeared of Burns (1951) and of Bishop (Bishop and Clare,

1953), testifying to the fact that this subject had attracted the attention

of a number of English and American electrophysiologists.

Certainly the method of stimulation of the cerebral cortex by

means of electrodes set on its surface has many deficiencies. First of

all, it is quite far from natural conditions of its stimulation; then,

at electrical stimulation of the cortical surface, simultaneous

excitation certainly occurs of many neuronal elements which are not

excited simultaneously during normal activity of the cortex. Thus,

it is possible to think that the bioelectrical reactions thereby

registered do not reflect normal activity of the cortical elements.

However, it is demonstrated that this method gives an opportunity for

the clarification of certain questions of the physiology of the cerebral

cortex. There is nothing unexpected in this, because with the aid of

the method of the electrical stimulation many important facts have been

obtained which have not lost their significance (Fritsch and Hitzig,

1870; Vvedenskii, 1897; Ukhtomskii, 1911, et al.). By the way, to

Pavlov belongs the idea of studying the action of direct electrical

stimulation of the various points of the surface of the cortex for

effects of conditional stimulants (1926).

A description will be given further on of the numerous

experiments with electrical stimulation of the cerebral cortex and

of the registration of the bioelectrical potentials thereby arising,

beginning with comparatively simple experiments and ending with those

quite complex in set-up and results.

The experiments were made on cats under nembutal narcosis

(25-40 mg. per kg. of weight). The operation consisted of exposing

the larger (cerebral) hemispheres of the brain; the dura mater was

removed directly before beginning the experiments. The temperature

of the surrounding air was 30-33°C.

Electrodes. Steel or silver needles served as stimulating

electrodes. For discharge of the biopotentials from the cortical

surface silver ones served for electrodes. For discharge from the

various layers of the cortex in the first experiments steel needles,

sealed with lacquer to the tip, were used. In subsequent experiments

discharge was effected with thin electrodes made of sealed constantan

(a nickel-copper alloy) wire, about 80 microns in diameter. Discharge

electrodes were fixed with microscrews to the cranium.

In experiments in which simultaneous discharge of the biopotentials

from two points of the surface of the cortex was required a special

electrode holder was used, which presented a plate of plexiglass 7 x

8 cm. in size. Into the plexiglass plate 49 openings were drilled with

thread (of screw). After fastening this plate in the holder, screwed

to the frontal bone, the discharging and stimulating electrodes were

screwed into the openings found over the points of the cortex involved.

The electrodes were installed in the following way: to the silver wire

with a thickened part of the end was soldered a flexible isolated

conducting wire on which are wound several loops of isolation tape.

The electrode was inserted into a metallic tube with thread (of screw),

which was screwed into the opening in the plexiglass. With the help

of the electrode holder described it was possible to arrange quickly

and with great accuracy several stimulating and discharge electrodes

over the surface of the brain.(Footnote: Recently Dawson (1954b)

published a description of the electrode holder with electrodes extremely

similar to those designed by me (in 1950).)

Location of electrodes in experiments with sinking of the electrodes

into the brain was determined by movement of the microscrew. Moreover,
in a number of experiments the discharge electrodes were coupled so
that the tip of the first came within 0.4-1.5 mm. of the end of the
second. When at rotation of the microscrew this electrode was found
on the surface of the brain, the first entered into the brain to a
precisely determined depth. In a number of cases histological
investigation was made of this part of the cortex into which the
electrodes were driven. In preparations made by Nissl's method it
was possible to find the track left by the electrode. (Footnote:
Histological investigations were made by S. Beritashvili.)

Stimulation of the cortex was effected by bipolar electrodes
with 1.5 mm. interpolar distance. The irritating stimuli lasted 0.2
or 0.5 millisecond. A stimulus that lasted 0.2 millisecond reached
a height of 75% after 20 microseconds and the apex after 60 micro-
seconds. At frequency increase of the stimulation to 100 per second,
the amplitude of the stimuli was reduced 5% and their character was
not altered.

The bipolar method of stimulation was selected as a result
of the following. During unipolar stimulation under the electrode
located on the surface of the cortex there is a thick field penetrating
the cortex perpendicularly with a compact cone of electrical lines
into which the deep layers fall. At bipolar stimulation the field

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is far more compact in the region of the surface layers (Dusser de

Barenn, 1934b). Thus, if the objective is to stimulate the elements

of the deep layers, then it is more advantageous to choose unipolar

stimulation (as was done in the experiments of Dusser de Barenn and

Adrian). If the objective to be pursued is to stimulate surface

layers as isolated as possible, then it is more advantageous to use

bipolar stimulation.

In the conditions of my experiments one-minute stimulation of

the surface layers by stimuli 0.2 millisecond in length at a voltage

of 30 volts and a frequency of 70 per second did not cause any

appreciable irreversible morphological changes of the neuron elements of

the stimulated portion of the cortex (S. Beritashvili, 1952).

Discharge was "unipolar". A thick needle inserted into the

bone over the frontal air-sinus served as indifferent electrode. As

will be demonstrated, with such a method of discharge the difference

of potentials that is registered is stipulated by the neuron elements

placed in direct proximity to the "active" electrode (see Renshaw,

Forbes and Morison, 1940; Kornmüller and Schaeder, 1938; Bishop, 1950).

The preparation was grounded; the grounding of the preparation did

not reflect appreciably on the character of the bioelectrical effects

being registered (see Gardner and Morin, 1953).

Intensification and registration. The biopotentials were

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intensified by boosters of alternating current with balanced entry,
i.e. very prolonged electrical fluctuations, if they arise in the
cortex, could not be recorded and/or distorted (see Kogan, 1949;
Beritov, Kvavilashvili and Roitbak, 1950). Recordings were made
with a two-ray cathode oscillograph. A series of experiments were
made with the use of a booster with a very great time constant.
A three-ray string oscillograph served for recording.

A description of the apparatus used, the schemes and charac-
teristics of the boosters of the alternating and direct current, the
deficiencies of the booster apparatus, the possible sources of errors,
the capacity for photorecording and for the marking of time, the
schemes of the relaxation stimuli, the general schemes of the layout
of apparatuses, etc. - all these are given in detail in the published
papers of Kvavilashvili (1945, 1950) and in Beritov, Kvavilashvili,
and Roitbak's article (1950), so I do not think it necessary to cite
these data in the present work.

Scheme of stimulation and discharge. With the usual layout of
the stimulating and discharging electrodes on the cortex, stimulation
by even the weakest currents causes "driving in" (or "stopping up")
of stimulus: the lamp is locked, i.e. the colossal voltage that
arises because of the polarization of the tissue falls on the mantle
(of the lamp). Polarization currents, that of one direction being

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under one pole and that of the other being under the other pole, arise

around the poles of the stimulating pair for a large territory. Their

amplitude and length are so considerable that if the oscillograph too

is not stopped up, then they cover up the biological effect.

One might think that on the cortical surface points could be

found in which the catelectrotonus and anelectrotonus neutralize one

another, as in the nerve there is a portion indifferent in regard to

the electrotonus between the C and A direct current. Actually this

can be discovered from the start on brain treated with formalin.

On living brain the problem proved more difficult because of the

complex and variable conditions of moisture, blood supply, and other

conditions affecting the character of the shunting of the current.

When the discharge electrode lies on the cortex at an equal

distance from each of the two stimulating electrodes, then at switching

on the stimulating current the booster usually is not shut off and

in response to the stimulating certain electrical potentials are

registered that change their character at boosting and increasing the

frequency of the stimulation and at changing the direction of the

stimulating current.

In Fig. 6 are presented a series of oscillograms showing the

results of such experiments. The stimulation and discharge electrodes

are placed on the sigmoid convolution; the distance from the discharge

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electrode to each of the two stimulating electrodes equals 1.5 mm.
With an intensity of stimulation of 2 volts and a frequency of 12 per
second the following effect is registered (osc. A); after a quick
variation of the ray, which is caused by the stimulating impulse
itself, a slow potential follows, which quickly reaches a maximum
and then almost exponentially dies out. At 4 volts (osc. B) the
amplitude of the potential is increased; its character remains the
same. This potential expresses itself mainly by a polarization of
the cortex. The discharge electrode in the case in question was
found under the dominant influence of the cathode, i.e. it recorded
the catelectrotonus. The fact that this is mainly the polarization
potential is demonstrated by the fact that at change of direction
of the stimulating current the potential changes its direction
(osc. B_1), but with this the complete symmetry of the polarization
potentials of the opposite sign, so characteristic at polarization
of the nerve fiber or of the nerve trunk, is lacking.

At boosting the stimulation to 8 volts (osc. C) an additional
potential,/altered polarization potential appears: superimposed on
the catelectrotonus is a double fluctuation, owing to which the total
length of the electrical potential is increased. At change of
direction of the stimulation current (osc. C_1) the anelectrotonus
which arises also is represented by a potential, the direction of

which is the same as with the catelectrotonus. Thus, the additional

potential that appears at a certain intensity of stimulation, super-

imposed on the polarization potential, in distinction from the latter

does not change its sign at change of direction of the stimulating

current.

At increasing the frequency of the stimulation to 25 a second,

the character of the potentials is changed by the course of the

stimulation: the supplementary potential considered gradually weakens

and then disappears; the polarization potential is left in pure form

(osc. D and D_1). Thus, the additional potential is a bioelectrical

reaction. It is possible to judge its form, amplitude, and length if

the polarization potential is deducted from the overall potential

(biopotential + polarization potential). The character of the latter

(at a given intensity of stimulation) can be concluded through the

potential which remains after prolonged stimulation of relatively

high frequency when the biopotential ceases to be provoked, evidently

from exhaustion of the nerve elements.

At increase of the distance between the discharging and stimulating

electrodes the amplitude of the potentials being registered, both the

polarization and the biopotential, is reduced. For instance, in Fig. 6,

E, potentials are shown discharged at intense stimulation at a distance

of 8 mm. from the point stimulated (the discharging electrode in this

case was in the region of another (cerebral) convolution).

(Legend to Fig. 6, textpage 28: (TN: Here, as in the preceding paragraphs, I have used the English alphabetical sequence from A-F for the illustration which thus read downward from left to right: A, B; B_1, C; C_1, D; D_1, E; and F.) Polarization and local potentials which arise at electrical stimulation of the cortical surface. Cat No. 7, July 4, 1949. Deep nembutal narcosis. Stimulating (Ag-AgCl) and discharging electrodes were placed on the surface of the gyrus sigmoideus post. Distance between them equaled 1.5 mm. A is an intensity of 2-volt stimulation. B is of 4 volts. B_1 is of 4 volts and the other direction of the stimulating current. C is of 8 volts. C_1 is of 8 volts and the other direction of the stimulating current. D is the beginning of frequent stimulation (25 per second), 16 volts. D_1 is 1 minute after stimulation. E is the discharge electrode placed on the gyrus suprasylvius at a distance of 8 mm. from the stimulating electrodes; the intensity of the stimulation is 16 volts. All these experiments were made without the use of a compensator. F are the polarization potentials balanced by means of a compensator. The biopotentials are discharged from a point 4 mm. distant from the stimulation electrodes; stimulation intensity 8 volts. Oscillation upward denotes negativity under the active electrode. Voltage and time designated for these illustrations: 1 millivolt and 20 milliseconds.)

The recording illustrations shown are essentially like those
which were given in works devoted to recording local potentials in
a nerve fiber (Hodgkin, 1938) and in the nerve trunk (Katz, 1947).
However, certainly the conditions of polarization in the brain are
incomparably more complex, but arising at direct stimulation of the
brain the biopotentials do not have their origin in the nerve fibers
of layer I because their length is very great as compared with the
length of the local potential in the nerve fibers.

As seen from the recordings presented and as other such
endeavors have shown, it is very difficult to record a biopotential
in pure form when the "neutral" point is found by transposing the
electrode from place to place. Usually it fails to be rid of a
considerable residue of unbalanced polarization potential. The
electrical intermediate (neutral) point far from always coincides
with the geometric, and complete compensation has succeeded in being
reached by means of a compensator. In Fig. 7 is presented the scheme
of stimulation and discharge used in the present investigation.
(Fig. 7, textpage 30: Scheme of experiment set-up for registration
of bioelectrical potentials arising in the neuron elements of the
cortex at its direct electrical stimulation. Ct. = (our St.) stimulator.)

In Fig. 6, F, are shown biopotentials not altered by polarization,
which were discharged at a distance of 4 mm. from the stimulating

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electrodes. Stimulation and discharge were carried out according to

the above-mentioned scheme.

1. Negative Potentials

At stimulation of the surface of the cerebral cortex of deeply

narcotized cat with electrical stimuli 0.2 millisecond in length and

at a distance of several millimeters in circumference from the point

of stimulation it is possible to register the bioelectrical potentials.

Comparatively intense stimulations must be used for their provocation:

the threshold of provocation of a biopotential with the stimulation

conditions in question usually equals 3-4 volts. (Footnote: With the

length of the irritating stimuli at 0.5 millisecond the threshold can

be lowered to 1 volt.) The threshold of stimulation of the sciatic

nerve for provocation of the cortical bioelectrical reaction is usually

less than 1 volt. Perhaps this is explained by the fact that the nerve

is stimulated at an interpolar distance equal to several millimeters,

and the brain was stimulated at an interpolar distance of 1.5 mm. In

special experiments with stimulation of the nerve-muscle preparation

of frog it was shown that at reducing the interpolar distance below 3 mm.

the thresholds of stimulation are increased because of the shunting effect

of the tissue fluid or of the physiological solution between the electrodes

(Beritov, 1930). It is necessary to think that high thresholds of

provocation of the cortical bioelectrical reaction are connected with

the shunting of the stimulation current. On the other hand, there
evidently also occurs a shunting of the answering biocurrent (Bishop
and Clare, 1953). Technical difficulty in recording the biopotentials
of the cortex in answer to its direct stimulation, of which we spoke
above, was aggravated by this circumstance, i.e. by the necessity
of using intense stimulations (up to 30 volts).

The effect of a single stimulation. With deep narcosis one
shock of stimulation or the first shock of rhythmical stimulation
causes a negative slow fluctuation of the bioelectrical potential;
after the negative fluctuation a weak positive one may follow.
Minimal length of a negative potential equals 10 milliseconds (Fig.
8, textpage 31: Bioelectrical potentials registered from the cortical
surface near the point stimulated. Cat No. 10, Oct. 24, 1949.
Nembutal. Discharge and stimulating electrodes are arranged on the
surface of the gyrus suprasylvius; the discharge electrode is found
at a distance of 1.5 mm. from the stimulating electrodes. The
intensity of the stimulation is 30 volts (the threshold 8 volts).
The effect of the first two shocks of stimulation with a frequency
of 16 volts (per) second (see Fig. 12, B). The first shock of
stimulation causes a simple negative potential with a length of
approximately 10 milliseconds; the second shock causes a more complex
effect: a series of additional negative fluctuations arise, designated

by arrows (2,3). Conditions: 0.5 millivolt, 5 milliseconds graphically
indicated.). The potential quickly, after 2 milliseconds, reaches its
peak and then falls. Its amplitude depends on the functional state of
the cortex and can reach 1-1.3 millivolts. During deep narcosis these
potentials are registered at a distance no greater than 5-8 mm., the
amplitude of the potentials being registered gradually diminishing
(Fig. 11) at moving the discharging electrode away from the stimulating
electrodes.

The following circumstances show that these potentials are
biological potentials stipulated by the activity of the cortical
elements, and not the polarization ones.

1) At shifting the direction of the stimulating current the
character of the effect is not changed.

2) During deep narcosis potentials are not registered from
other convolutions (of the brain) even at a most proximal distance
from the part being stimulated (Fig. 6). These two circumstances
were already indicated by Adrian (1936).

3) They cease to be provoked 15-60 seconds after the heart
has stopped. Chang (1951) demonstrated that during anoxia, which
was caused by the animal's having breathed pure nitrogen, they
disappear after 1.5 minutes.

4) At reducing the temperature of the cortex below 28°C. and

at increasing it above 40°C. their amplitude is reduced; they cease

to arise with the temperature below 22°C. and above 50°C. (Chang,

1952).

5) They are intensified during local strychnine poisoning (a

0.1% solution) of the cortex under the discharging electrode (Fig. 10,

textpage 33: Changes of the bioelectrical potentials provoked by

stimulation of the cortex after local strychnine poisoning of the

cortex under the discharge electrode. Cat No. 35, Nov. 13, 1950.

Continuation of experiments carried out in preceding illustration.

Stimulation and discharge electrodes were changed to another area

of the gyrus suprasilvius. Distance (between stimulating electrodes

and discharging electrode) = 6 mm. A is the effect of 30-volt

stimulation, 10 per second before poisoning. After this, strychnine

(0.5% solution) was applied to the brain under the discharge electrode;

the poison was removed after 2 minutes. B is the effect of the same

stimulation 3 minutes after the moment of poisoning, C is after 14

minutes, and D is after 20 minutes. After this, strychnine (0.5%

solution) was applied to the brain under the stimulation electrodes;

the poison lay there for 2 minutes. E is the effect of the same

stimulation (30 volts, 10 per second) 3 minutes after poisoning. F

is after 40 minutes. Conditions indicated in illustrations: 20

milliseconds, 0.6 millivolt. (TN: As before, in translation the

designations are interpreted with English alphabetical sequence; thus
by lines: A; B; C and D; E and F.))

6) They often cease to be provoked during local poisoning of
the cortex under the discharge electrode or under the stimulation
electrodes by a concentrated solution of strychnine, and these
disturbances are reversible (Fig. 9, textpage 32: Changes of bio-
electrical potentials provoked by electrical stimulation of the
cortex in consequence of local strychnine poisoning of the stimulated
part of the cortex. Cat No. 35, Nov. 13, 1950. A and B are of the
gyrus suprasylvius of the left hemisphere. Distance between stimu-
lation electrodes and discharge electrode is 6 mm. A is the effect
of stimulation with a frequency of 5 per second (30 volts) prior
to poisoning. After this, a ball of cotton soaked in a saturated
solution of strychnine nitrate was applied to the brain under the
stimulating electrodes. The strychnine was removed after 1.5 minutes,
the brain was dried at this place and washed with physiological
solution, and the stimulation electrodes were placed at the previous
site. B is the effect of the same stimulation 2 minutes after applica-
tion of strychnine to the cerebrum under the stimulation electrodes.
The effects were reduced 30 minutes after taking off the strychnine.

C and E are of the gyrus suprasylvius of the right hemisphere.
Distance between the stimulation electrodes and the discharge electrode

is 6 mm. C is the effect of stimulation (30 volts, 5 per second) prior

to poisoning; after this the area under the stimulation electrodes

was poisoned with a 0.5% solution of strychnine; the strychnine was

removed after 3 minutes. D is the effect of the same stimulation 3.5

minutes after application of the strychnine to the brain under the

stimulation electrodes. E is the effect of the same stimulation 15

minutes after the recording of D. Indication of 0.3 millivolt for A,

of 0.6 millivolt for C.). In the experiments of Beritov and Roitbak

(1950b) on spinal cord of frog it was discovered that at relatively

great concentration of strychnine (general or local poisoning) it

is possible to observe two stages of its effect: at first, the

posterior and anterior root potentials attenuate, and only after

several minutes do they begin to intensify. Thus, weakening of the

cortical negative potentials after strychnine poisoning should be

ascribed to its parabiotic action on the neuron elements. Histological

investigation of the part of the cortex subjected to poisoning by

saturated solution of strychnine showed drastic morphological changes

of the cortical neurons (S. Beritashvili, 1952). In connection with

what has been mentioned above, it is possible to set forth the following

fact obtained by Chang (1951): the negative potential adjusted to be

provoked 8 min. after a strip of paper saturated with a 2% solution of

cocaine was placed on the cortex between the stimulation and the

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discharge electrodes. In analogous experiments of Burns and

Grafstein, 1952, it ceased to be recorded after 4.8 minutes. In

my experiments the bioelectrical potential ceased to be provoked

2 minutes after poisoning of the cortex under the discharge

electrodes by a saturated solution of strychnine.

With a frequency stimulation of 25-50 per second the

potentials considered quickly attenuate (see below).

Effects similar in character arise at stimulation of any

part of the dorso-lateral surface of the cortex, i.e. the character

of the effect is not appreciably altered in areas of the cortex

different according to functions.

At powerful stimulations and at repeated stimulations addition-

al negative fluctuations can arise (Fig. 8), but this is not charac-

teristic to effects during deep narcosis and will be specially

considered in a subsequent part of this chapter.

At a distance of 1.5-2 mm. between the stimulation electrodes

and discharge electrodes the negative potential sets in 2-2.7

milliseconds after the moment of application of stimulation. At

removing the discharge electrode, together with reduction of the

amplitude of the potential the latent period of its arising is

increased and its character changed: it reaches the peak more

slowly. Increase of the latent period cannot always be detected

because of the fact that the interval between the moment of stimulation

and the moment of the arising of the biopotential is masked by an

unbalanced residue of polarization potential (this took place in

registration A, Fig. 11). (Legend to Fig. 11, textpage 34: Decrement

spread of negative potentials. A is cat No. 22, May 12, 1950. Bio-

currents are discharged simultaneously from a point of the surface of

the gyr. sigmoideus post. at a distance of 1.5 mm. from the stimulation

electrodes (upper curve) and from a point at a distance of 3 mm. from

the stimulation electrodes (lower curve). Intensity of stimulation

25 volts, frequency about 10 per second. B is cat No. 7, July 4, 1949.

Gyr. sigmoideus post. Distance between stimulation electrodes and

discharge electrodes 2.5 mm.; the effect of one shock of stimulation

(8 volts). C is the effect of a single stimulation shock (8 volts)

after the discharge electrode was shifted a distance of 5 mm. from

the stimulation electrodes. D and E are of cat No. 37, Jan. 7, 1951.

Slight narcosis (6 hours after nembutal injection). Stimulation

electrodes P_1 are placed at the posterior pole of the gyr. suprasylvius;

at a distance of 5 and 11 mm. from them on the surface of this convolution

are placed discharge electrodes E_1 and E_2; the second stimulation P_2

pair are placed on the surface of the gyr. sigmoideus post, at a distance

of 2 mm. from E_2. In Fig. F is given the arrangement scheme of stimula-

tion and discharge electrodes. The biocurrents are discharged

simultaneously from point E_1 (upper curve) and E_2 (lower curve). D

is stimulation carried out through P_1 electrode, frequency 16 a second

(25 volts). E is stimulation effected through R_2 electrodes, frequency

of stimulation 12 per second (25 volts). Indications for A: 0.3

millivolt, 20 milliseconds.) In recordings B and C in Fig. 11 (right

upper third, left and right respectively) the effects are shown of

stimulation of the gyr. sigmoideus. In experiment B the discharge

electrode was 2.5 mm. from the pair of stimulating electrodes; the

latent period of the arising of the biopotential equaled 3.3 milliseconds.

In experiment C the distance between the points of stimulation and of

discharge equaled 5 mm.; the latent period of the arising of the

biopotential equaled 8 milliseconds. If the rate of spread is calculated

on the basis of the differences of latent periods and of the distances

in experiments B and C, then the magnitude reached is approximately

0.5 m. per second. Thus, if it is assumed that the activity is in any

way spread from the point of stimulation to the point of discharge,

then the rate of this spread, on the basis of the above-mentioned

experiment, is of the order of 0.5 m. per second, which agrees with

Dow's data (1949) in respect to the spread rate of potentials in the

cortex of the cerebellum. Chang thus found that the spread rate of the

negative potentials considered in the cortex of cat equaled 1 m. per

second (1951).

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According to Adrian and Chang, the negative bioelectrical potential discharged from the cortical surface of cat at its stimulation is not registered at a distance greater than 5 mm. from the stimulation electrodes and is not registered from another convolution even if the distance from the site of stimulation is very small. The recordings presented in Fig. 11, D and E (lower 2/3 on the left, upper and lower, respectively), contradict these two positions. The set-up of the experiments was the following: on the surface of the cortex of cat under relatively shallow narcosis were placed 2 pairs of stimulating and 2 discharge electrodes (see Fig. 11, F (lower 2/3 at the right); the first stimulating pair (P_1) and the first discharge electrode (E_1) are established on the posterior portion of the gyr. sigmoideus and the second stimulating pair (P_2) and the second discharge electrode (E_2) are placed on the gyr. suprasylvius. The distance P_1 - E_2 = 11 mm. At stimulation through electrode P_1 (experiment D) and electrode P_2 (experiment E) negative potentials are registered in both convolutions; their amplitude is less at the more remote point. Thus, negative biopotentials at stimulation of the cortical surface can arise at a considerably greater distance from the place of stimulation than was estimated. They may arise in another convolution. We have thus been confronted with new facts, which will be considered in detail in the following paragraphs of this chapter.

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As for the rate of spread of activity in experiments D and E,
Fig. 11, it is of the order of 0.85 m. per second and it is identical
during the spread from the dyr. suprasylvius into the gyr. sigmoideus
(P_1 stimulation) and during spread from the gyr. sigmoideus into the
gyr. suprasylvius (P_2 stimulation). Attention is attracted to the
fact that the rate of spread in the case in question is greater than
in experiments B and C and this can perhaps be connected with the
different depth of narcosis in these two cases.

According to Burns' data (1951), in isolated part of non-
narcotized cortex[*] direct electrical stimulation of its surface
causes the same negative potential as described for narcotized animals.
Its rate of spread equals 2 m. per second. Thus, the rate of spread
of activity here is even greater in experiments with non-narcotized
cortex. ([*]Isolated from subcortex and from the rest of the cortex,
a strip of gyr. suprasylvius of cat, 20 mm. in length, was kept
connected with the rest of the cortex only by the blood vessels.)

The fact that at increase of distance between the stimulation
electrodes and the discharge electrode the latent period of the
bioelectrical reaction being registered is increased is demonstration
that extremely local biopotentials arise at direct electrical
stimulation of the cortex. The latent period would not be increased
with increase of the distance between the stimulating and the discharging

electrodes if the potentials arising are spread simply physically
with decrement. Thus, the region of the discharge of these biopotentials
is limited practically to the region of their arising.

As known, during investigations of the cortex by the method of
its direct electrical stimulation room for doubt has always been left
as to whether or not one or another external effect to be observed at
stimulation of the cortex is due to shunting of currents to other
proximate parts of the gray or white matter (Vvedenskii, 1897;
Ukhtomskii, 1911). The fact that the latent period of the cortical
biopotential is increased at moving the discharge electrode away
shows that with the conditions of stimulation in question the bio-
electrical potentials being registered are stipulated by stimulation
and excitation namely of that point of the cortex to which the
stimulating electrodes are applied, i.e. shunting of the stimulating
current does not occur, at least to such a degree, in order that at
a distance of 2-3 mm. these loops of current could show such a
stimulating effect on the neuron elements as would lead to the
arising of the potentials being considered. Thus, the area of
indirect provocation of neuronic elements of the cortex has been
limited practically to the region of spread of the stimulating elec-
trodes.

On the other hand, the fact considered (length of latent period

in connection with the moving away of the discharge electrode) shows

the accuracy of the oscillographic method being used. The discharge

electrode shunts precisely the bioelectrical activity of that part

with which it comes in contact. This is a good argument against

those electrophysiologists who think that the potentials discharged

are always the expression of the sum total of a great number of

potentials resultant from the bioelectrical activity of hardly the

whole of the cortex and that at excitation of any part of the cortex

potentials, purely physical, can be discharged from remote parts of

it, etc.

Very persuasive positions have been exposed on the extraordinary

local character of stimulation and discharge by experiments with

strychnine poisoning of the parts being stimulated and subject to

discharge which were spoken of above. During local point posioning

under stimulating or discharge electrode the bioelectrical effects

that arise at stimulation can temporarily cease being registered.

When this occurs as the result of poisoning under the stimulating

electrodes (Fig. 9), this shows the extremely local character of

the stimulation: the loops of current do not stimulate parts of the

cortex found at a distance of several millimeters beyond the poisoned

point. When this occurs as the result of poisoning under the discharge

electrode (Fig. 10), this shows the extreme local character of the

discharge of biopotentials: the discharge electrode does not discharge

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biopotentials from a portion of the cortex found beyond the poisoned
point, from parts in which biopotentials of greater amplitude arise
than those arising normally under the dis charge electrode because
they are found closer to the site of stimulation.

The facts obtained oblige referring quite skeptically to the
possibility of a diffuse and territorially spread physical influence
of the biopotentials that arise during excitation of any complex of
neuronic elements, for example to the possibility of the diffuse
anelectrotonic influence of the potentials through tissue fluid on
a great number of surrounding neuronic elements, as this was proposed
earlier by Beritov (1937b, 1948) and by Beritov and Roitbak (1948b),
or to the possibility of the anelectrotonic or catelectrotonic effect
of currents of the granular layers of the cortex, as was considered
probable by Ukhtomskii (1939-40).

The effect of rhythmical stimulations of the cortex during
deep narcosis. (Legend to Fig. 12, textpage 37: Negative potentials
caused by electrical stimulation of the surface of the cortex with
different frequency of stimulation. Cat No. 10, October 24, 1949.
Nembutal. The discharge electrode and the stimulation electrodes
are placed on the surface of the gyr. suprasylvius; the discharge
electrode is a distance of 1.5 mm. from the stimulating electrodes.
Intensity of stimulation 30 volts (threshold 8 volts). A is a
stimulation frequency of about 3 per second; B is 16 per second; the

B_1 are effects after 1 minute of stimulation. C is frequency of

stimulation of about 50 per second. C_1 is after 30 seconds of

stimulation. D is frequency of stimulation of 100 per second. E

is the discharge electrode removed a distance of 2.5 mm. from the

stimulating electrodes; frequency of stimulation at 15 per second

is instantaneously switched to 100 per second; E_1 is after one

minute of stimulation with a frequency of 100 per second, and

change-over occurs to a frequency of 15 per second (Roitbak, 1954a).

20 milliseconds and 1 millivolt indicated.) During a stimulation

frequency of 3 per second the subsequent shocks cause greater

effects than the first (Fig. 12, A). With a stimulation frequency

of 10-20 per second growth of effects occurs for the first 0.2-0.5

second of stimulation (Fig. 12, B); furthermore, the effects are some-

times complicated by additional waves arising. At stimulation

frequencies of 50-100 per second the effects progressively and quickly

attenuate and in the course of the first 0.2-0.5 second tetanization

can dwindle to nothing (Fig. 12, C-E).

处When the functional state is poor, stimulation of the cortex

provokes negative potentials of little amplitude. With a stimulation

frequency of 10-20 per second the character of effects in the course

of stimulation is not changed, i.e. the phenomenon of growth in

amplitude of the potentials is lacking (Fig. 13, textpage 38: Bio-

electrical potentials that arise in response to stimulation of the

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cortex during deep narcosis. Cat No. 4, June 6, 1949. Deep

narcosis after injection of a double dose of nembutal. Stimulation

and discharge electrodes in the gyr. sigmoideus post.: distance

between them 3 mm. Intensity of stimulation 30 volts. A is

frequency of stimulation at 8 per second. B is 15 per second. C

is 40 per second, and C_1 is after several seconds of stimulation.

D is frequency of stimulation at 80 per second. 20 milliseconds

and 0.6 millivolt indicated.). At frequency of stimulation of 50

per second the effects quickly dwindle to nothing; at a frequency

of 100 per second only the few first shocks cause appreciable

bioelectrical potentials.

In Fig. 14, A, an experiment is set forth with cerebral

stimulation at a frequency of 25 per second. At first the magnitude

of the effects grows, and after the fourth stimulation shock the

effects progressively attenuate. The twentieth stimulation shock

causes a three times weaker potential than the second. In experiment

B the frequency of stimulation is momentarily increased to 125 per

second. After slight fluctuation caused by the first shock of

tetanic stimulation the subsequent shocks produce no effect. In

recording C after 2.7 seconds of tetanization at a rhythm of 125 per

second the frequency of stimulation is again shifted to 25 per second;

thereby effects arise the same in amplitude as prior to application

of stimulation at 125 per second. Thus, these 2.7 seconds of tetanization

have influenced little the character of the effects of the infrequent

stimulation. In recording D after 3 seconds of stimulation at a rhythm

of 25 per second when the effects have considerably attenuated a shift

of frequency of stimulation to 125 per second was made, and then after

a stimulation of several seconds at a rhythm of 125 per second the

frequency was again changed to infrequent. The effects of infrequent

stimulations after this intensified approximately four times, and

these intensified effects lasted rather long. Thus, the impression

is created that partial repose of the cortical elements being activated

has occurred after a period of tetanization of the cortex.

(Legend to Fig. 14, textpage 39: Bioelectrical potentials

provoked during different conditions of stimulation of the cortical

surface. Cat. No. 26, June 3, 1950. Stimulating electrodes and

discharge electrode are placed on the surface of the gyr. suprasylvius;

distance between the discharge electrode and the stimulating electrodes =

2.5 mm. Intensity of stimulation 30 volts. A=frequency of stimulation

at 25 per second. B - a continuation of recording A; the frequency of

stimulation is momentarily switched from 25 per second to 125 per second.

C - termination of 2.7-second tetanization at 125 per second and

change to a stimulation frequency of 25 per second. D - after 3 seconds

of stimulation at a frequency of 25 per second a shift is made to a

frequency of 125 per second. E - after 15 seconds of tetanization
at 125 per second the frequency of stimulation is changed to 25 per
second. F - the effect of the application of 30-volt stimulation
at 20 per second after 1 minute of repose (Roitbak, 1954a). Indications
of conditions on the illustrations 20 milliseconds and 0.4 millivolt.)

Thus, we run into the following phenomena. First, we encounter
exhaustion when the relatively infrequent stimulations at prolonged
stimulation begin to give gradually attenuating effects. Secondly,
we run into sharp attenuation or absence of effects when there is
an increase of frequency of the stimulation to 50-100 per second.
This is not an expression of exhaustion, because at lengthening the
interval of stimulation an effect arises immediately (see also Fig.
12, E), i.e. we apparently run into phenomena of the worst-best
("pessimum-optimum") order. In the third place, we run into "repose"
at the time of prolonged tetanic stimulations, but also, as in the
experiments of Vvedenskii, the present repose is much more effective
than "repose" at the time of the worst ("pessimum") tetanization
(Fig. 14, F).

At consideratinn of these facts obtained during direct
electrical stimulation of the cortical surface analogy intrudes
with the "pessimum-optimum" phenomena studied by Vvedenskii in
muscle-nerve preparation (1886) and in spinal cord (1904). However,

we later perceive that the correctness of such an analogy can be

confirmed with doubt.

Potentials discharged from different layers of cortex at

stimulation of its surface. Below are mentioned the results of

experiments with discharge of potentials from different layers of

cortex during electrical stimulation of its surface.

In Fig. 15, A and B, are presented electrical effects

discharged from the surface of the cortex at a distance of 2 mm.

from the stimulating electrodes at different intensities of

stimulation. At 12 volts (osc. A) negative potentials arose

with an amplitude of about 1 milliv.; at 30 v. the amplitude of

the potential exceeded 1.5 milliv. and an additional negative

fluctuation arose (osc. B). Then the discharge electrode was

sunk more deeply into the cortex; histological investigation

showed that the end of the electrode was in layer V. In Fig.

15, C-E, are presented the electrical effects discharged from

layer V during different conditions of stimulation of the cortical

surface (the position of the stimulating electrodes was not changed).

At 6 volts the effects were not provoked, whereas from the surface

with this threshold intensity of stimulation considerable negative

potentials were discharged; at 12 v. insignificant positive

fluctuations were discharged from the depths (osc. C); at 30 v.

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from layer V considerable positive potentials were discharged with

an amplitude of 0.75 millivolt (osc. D). At tetanic stimulation

with a rhythm of 50 per second a long positive oscillation was

obtained, after which a negative arose and then the ray came back

to the abscissa (osc. E); at cessation of stimulation a slow two-

phase fluctuation also arose. Analysis of this curve shows that

throughout the whole time of stimulation a long, constantly attenuat-

ing, positive potential occurred (see Roitbak, 1950a). After these

experiments the electrode was placed on the surface in line with

the place of puncture. In response to stimulation negative potentials

again arose of somewhat lesser amplitude than those discharged prior

to deep placement of the electrode (osc. F and G). This indicates

that the puncture does not damage the cerebral tissue to any

considerable extent, as was established by experiments on spinal

cord (Beritov and Roitbak, 1948a). At stimulation with a frequency

of 50 per second it is necessary to assume the arising of a long

negative fluctuation (osc. H).

(Legend to Fig. 15, textpage 41: Bioelectrical potentials

discharged from the surface and from the depths of the cortex at

stimulation of the cortical surface. Cat No. 16, Feb. 11, 1950.

On the surface of the gyr. suprasylvius are placed stimulating

electrodes and at a distance of 2 mm. from them a discharge

microelectrode. The provocation threshold of the biopotentials

is 6 v. A is 12-v. intensity of stimulation, and the beginning of
stimulation is at a frequency of 10 per second. B is intensity of
stimulation of 30 v. C is discharge electrode sunk by means of a
microscrew to layer V; intensity of stimulation 12 v., frequency
10 per second. D is intensity of stimulation 30 v. E is beginning of
brief tetanic stimulation during a frequency of 50 per second (30 v.);
E_1 is end of stimulation. F is microelectrode raised and placed on
the surface of the brain, intensity of stimulation 12 v., frequency
10 per second. G is intensity of stimulation 30 v. H is beginning
of brief tetanic stimulation at frequency of 50 per second; H_1 is
end of stimulation. Condition indicated on illustration at G is
1.2 millivolts.)

(Legend to Fig. 16, textpage 42: Bioelectrical potentials
discharged from the surface of the cortex, from the depths of the
cortex, and from the white matter at stimulation of the surface of
the cortex. A is for cat No. 15, Feb. 8, 1950. Biopotentials are
discharged by microelectrodes from the surface of the brain (upper
curve) and from a depth of 0.7 mm (lower curve); gyr. suprasylvius,
its posterior pole. Stimulating electrodes are placed on the surface
of the brain at a distance of 1.5 mm. from the microelectrodes.
The intensity of stimulation was 25 v., the frequency 10 per second.

B-D is for cat No. 27, June 7, 1950. Biopotentials discharge

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from the surface of the cortex by the very fine electrode E_1 (upper curves) and from the depths by the needle-shaped electrode E_2 (lower curves). The stimulating electrodes are placed on the surface of the cortex. B is with E_2 sunk to a depth of 0.1-0.2 mm.; stimulating electrodes are found at a distance of 3 mm. from E_1; frequency of stimulation 40 per second (25 v.). C is with E_2 sunk down to the white matter; stimulating electrodes are found at a distance of 6 mm. from E_1; frequency of stimulation 40 per second. D is with a frequency of stimulation of 10 per second.)

In Fig. 16 recordings are presented that were obtained in experiments with simultaneous discharge of biopotentials from different layers of cortex, from the surface and from a depth of 0.5 mm. (osc. A), from the surface and from layer II (osc. B), from the surface and from the white matter (osc. C and D). On the basis of these experiments it is possible to make important factual conclusions: whereas from the surface of the cortex a negative potential is registered, in the different layers of the corresponding point of the cortex potentials different in sign are registered: in layers I and II there is a negative potential; at somewhat greater depth (0.5 mm.) no certain potential can be registered or a weak positive fluctuation is registered; in layers V and VI, as well as under the cortex from the white matter, a positive potential is registered, the negative potential

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discharged from the surface being in almost mirror-image form.

The "inversion" of the sign of the potential when the discharge
electrode is sunk down into the cortex occurs anywhere close to the
surface of the cortex, which shows directly that the neuronic elements
of the surface layers are the source of the negative potential
discharged from the surface of the cortex.

Adrian (1936) came to such a conclusion on the basis of the
fact that after thermocoagulation of the surface layers of the cortex
the negative potential in this part ceased to be stimulated. Bishop
and Clare (1953) in experiments with discharge of biopotentials of
the cortex simultaneously from three levels, from the surface layers,
the middle layers, and the white matter, found that at stimulation
of the surface of the cortex of the upper 1/3 of the cortex a negative
potential is produced and at stimulation of the lower 2/3 either no
potential is produced or the middle electrode is positive in respect
to the white substance.

Thus, from the facts to be had it follows that the negative
potential, discharged from the surface of the cortex at direct
stimulation of it, expresses excitation of the neuronic elements of
the surface layers of the cortex.

Histological information on layer I of the cortex. At
stimulation of the cortex by electrodes placed on its surface, the

stimulating current should primarily act on the nerve elements found
on the surface, i.e. on the elements of layer I of the cortex.

Information on layer I of the cortex, quite meager in ordinary
textbooks, can be found in the works of 1) Cajal (1893), 2)Bekhterev
(1898), 3) and 4) Lorente de No (1933, 1943), 5) Bliumenau (1925), 6)
O'Leary and Bishop (1938), 7) Zurabashvili (1947, 1949), 8) Sarkisov and
Poliakov (1949), and 9) Chang (1953).

Presented below are data on the structure of layer I, on the
derivation of the fibers of layer I, and on their distribution and
terminations. For brevity, literary references are designated by
the figures corresponding to the above-mentioned list.

At examination of cortical preparations stained by the Cajal
method or by Golgi's method the following well known,and, at the
same time, important fact is conspicuous: in layer I of the cortex
the predominant nerve fibers are those that proceed on a tangent with
the surface of the cortex; in layer II dendritic offshoots predominate
which arise from the pyramidal cells of the subjacent layers.

The fibers of layer I. Part of these fibers are devoid of
medullary membrane, others are myelinated and form thick tufts[2,3],
especially in the upper and lower levels of the layer[6]. The fibers
arranged in the deepest parts of layer I and on the boundary with
layer II were described by Bekhterev as a special layer of fibers.

Among the fibers of layer I is a certain number of thick fibers
proceeding for great distances[3]. To them apparently belong the
axons of the horizontal cells of layer I, the length of which is
so considerable that it is impossible to trace them to the end,
however large the section may be[5], even should it measure several
millimeters[8], and likewise axons of cells with the axon proceeding
from the subjacent layers of the cortex[3].

Fibers of the outer part of the layer have mainly a direction
diametrical to the length of the convolution, whereas the fibers
described by Bekhterev have a direction corresponding to the length
of the cerebral convolutions. According to Bekhterev, the first
serve as a connection for the two neighboring convolutions and the
second serve as a connection between the most diverse parts of one
and the same convolution, often more or less remote from one another[2].

Lorente de No finds that part of the thick fibers of layer I
do not go beyond the limit of a given cyto-architectonic field, others
come from adjacent fields[3].

Bekhterev came to the conclusion that "the first layer of the
cortex generally represents to the highest degree conditions favorable
to associated activity"[2]. O'Leary and Bishop, 40 years after Bekhterev,
considered it quite probable that the fibers of layer I participate in
the formation of association courses like certain courses in the white
matter.

Origin of fibers of layer I. The fibers of layer I are chiefly axons or collaterals of different cortical neurons; moreover, collaterals proceed into layer I of certain afferent fibers. The following kinds of fibers are found in layer I.

1) The fine collaterals of the associative and collosal fibers. (This was ascertained by Bekhterev, and this was confirmed by the most recent histological investigations[4,9].

2) Collaterals of the axons of the pyramidal cells of layers II-VI[2,3,6]. In the higher mammals the horizontal dendrites of the recurrent collaterals of the cells of the deep layers form in layer I sturdy tufts of myelinated fibers[4]. By the way, Bekhterev thought the collaterals of the cells of the subjacent layers the chief source of the layer of fibers discovered by him at the boundary of layers I and II.

3) Axons and collaterals of neurons with a short axon of cortical layers I-VI[3,6]. Concretely, in the formation of layer I the following kinds of neurons with short axon participate: a) cells with ascending axon of layer II; b) cells with ascending axon of layers III and IV; c) cells of layers III and IV, similar to the pyramidal, with ascending axon; the axon produces collaterals along the way; d) small cells with round forms of layers III and IV with axon rising into layer I, giving off collaterals along the way; e) cells of globular form of layers III and IV; the axon forms

a plexus around the cell, and the rising dendrite produces in layer
I several tangential fibers[3]; and f) cells of layer V with the
axon or its collaterals rising into layer I[6].

O'Leary and Bishop indicate two main types of neurons with
short axon in the granular layer (IV): neurons with rising axon
and neurons with descending axon; the axons of the first reach
layer I. Bliumenau attaches great importance to the fact that,
although cells with rising axon reach layer I, they are contained
in all layers of the cortex, but there is an especially great number
of them in layer IV. We shall come back again to this circumstance.

Dendrites of layer I. In layer I the ramifications are completed
of the top dendrites of the pyramidal cells of the subjacent layers.
According to Cajal, the fibers of layer I form a network in the
meshes of which the tops of the dendrites terminate, studded, as
Bekhterev also confirms this, with a great number of thornlike off-
shoots to which, after Sukhanov, Sarkisov and Poliakov attach
extraordinary importance, assuming that they serve for contact with
the synaptic terminals (Sarkisov and Poliakov, 1949; see also Chang,
1952). The top dendrites at having attained layer I, or somewhat
earlier, split into dendritic branches, assuming a horizontal direction;
the length of the horizontal dendritic branches does not exceed 2 mm.[9],
according to the data to be had.

Into layer I enter the dendrites of the tiny cells of layer II and the dendrites of the spindle-shaped cells of layers III and IV and ascending dendritic branches of certain neurons with short axon from layers III and IV, but likewise the top dendrites of the pyramidal neurons comprise the overwhelming mass of dendrites of layer I.

Synaptic connections of the system of fibers of layer I. It has been found that the thick long fibers of layer I, which are the rising axons of the cells of the subjacent layers, produce a great number of collaterals that ramify not only in layer I but also in layer II[3] and that the collaterals of the axons of the cells of layer I (of Cajal's horizontal cells) enter not only into layer II but even into layer III[6]. Nevertheless, Bekhterev concluded that the top dendrites "present to the highest degree favorable conditions for association" through contact with the branches of the axons that penetrate here. Cajal saw that the fibers of layer I, particularly the axons of the cells of layer I, terminate with ramifications in the top dendrites of the pyramidal (cells). Bekhterev presents an illustration (1893, p. 213) in which are shown the top dendrites and the terminations of the fibers of layer I on them. Zurabashvili found in preparations stained for the synapses (according to Hoff's modified method)

that there are numerous synaptic tufts to the top dendrites in layer

II and the whole layer is strewn with presynaptic fibers[7]. According

to Lorente de No, the top dendrites receive impulsation chiefly from

fibers of the layer I plexus and not of the layer II. According to

his data, the number of synapses for dendrites of the pyramidal cell

in layer I is 1000 times greater than in layer II[3]. Synapses

congregate chiefly in the area of the bifurcation of the dendrites

and in dendritic branches after bifurcation for dendritic offshoots

of the motoneurons of spinal cord of cat; there are fewest of them

in the part of the dendritic trunk immediately before its bifurcation[7].

It is possible to think that analogously for the top dendrites the

greatest number of synaptic terminals of the fibers of layer I is in

layer I in the region of their bifurcation and in their horizontal

branches.

On the basis of known neurological data in regard to layer I

of the cortex, it is possible to make the following conclusions.

1. In layer I there are medullated and unmedullated fibers

attaining great length. These fibers connect with one another the

adjacent convolutions, the different parts of a convolution, as well

as the different cortical fields.

2. The bifurcations of the fibers of layer I terminate

principally in layers I and II of the cortex. In layer I they form

numerous synaptic terminals in the top dendrites of the pyramidal

neurons. In layer II they participate in the formation of a complex
network of fibers terminating in the neuronic elements of layer II and,
among them, in the trunks of the dendrites of the pyramidal cells.

3. At stimulation of the fibers of layer I activation occurs
mainly of the dendrites in layer I of the cortex. Thus, in the
cortex of the larger hemispheres (cerebral) there are anatomical
reasons for the possibility of more or less isolated activation of
dendrites, a clarification of the function of which is the present
problem of neurophysiology.

4. Since the fibers of layer I proceed from the pyramidal
neurons of all subjacent layers of the cortex and from the cells with
axon rising from all subjacent layers of the cortex, then, consequently,
the top dendrites of the pyramids can be activated from a vast number
of sources; excitation of the neurons of any layer of the cortex can
be transferred through the system of the fibers of layer I to the
tops of the dendrites of the pyramidal neurons.

Origin of negative biopotentials discharged from the cortical
surface at direct electrical stimulation of it. It can be considered
established that the slow negative potential arising directly in
response to electrical stimulation of the surface of the cortex
expresses mainly the state of excitation of the dendrites of the
dendrites of the cortical surface layers and is a "dendritic potential".

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Beritov came to this conclusion in 1941 on the basis of the data

of Adrian (Beritov, 1941). Chang (1951), Eccles (1951), and Bishop

and Clare (1953) have arrived at the same view.

The facts obtained in experiments with stimulation of the

different layers of the cortex testify in favor of this, that the

arising of negative potentials at stimulation of the surface of

the cortex is connected with excitation of the elements of layer I.

Stimulation of the cortex was made by the "unipolar" method, by

impulses 0.5 millisecond in length, and by means of a glass microelectrode.

When the stimulation electrode was on the surface of the cortex or at

a depth of 0.1-0.4 mm., then during a certain intensity of stimulation

from the surface of the cortex in response to the stimulation a

negative potential was discharged. At sinking the electrode further,

i.e. at stimulation of the middle and deep layers of the cortex, from

the surface of the cortex was registered (at a given intensity of

stimulation) a negative potential of far less amplitude than at

stimulation of the surface layer of the cortex (Burns and Grafstein,

1952). Thus, the greatest activation of the top dendrites occurs at

stimulation of the surface layer of the cortex in which the system

of fibers of layer I is found. The arising of the negative potentials

during stimulation of the middle and deep layers can be explained by

the fact that stimulation thereby occurs of the collaterals and axons

that rise into layer I or of the corresponding cells of these layers,
i.e. by the fact that the system of fibers of layer I is again thereby
excited.

Slow negative potentials are recorded even during deep narcosis
at a considerable distance from the part of the cortex stimulated, the
latent period of their arising being more prolonged in the more remote
points than in the more proximal. The following fact can serve as
proof that the spread of these potentials through the cortex is
connected with the spread of the excitation by the elements of layer
I: after incision of the cortex between the stimulating electrodes
and the discharge electrode to a depth of 0.13 mm., the negative
potential ceases to be registered (Burns and Grafstein, 1952).

In layer I there are, as said, ramifications of the top
dendrites and of the system of the tangential fibers. The length
of the horizontal dendritic branches in layer I does not exceed
2 mm. (Chang, 1951); consequently, it is fitting to think that the
spread of the negative slow potentials occurs by means of the fibers
of layer I. The rate of spread of the slow potentials is determined,
thus, by the rate of spread of the excitation along the fibers of
layer I. It is known that the very fine fibers of the peripheral nerves
conduct the excitation at a rate of the order of 0.7 meter per second,
i.e. at a rate which approximates that with which the activity in the
surface layers of the cortex is spread during deep narcosis.

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The rate of spread is considerably altered in connection with
change of temperature of the cortex: at 29-32°C. it equals 2 m. per
second, at 26° 0.7 m. per second, and at 22° 0.4 m. per second (Chang,
1952).

Eccles in a survey article on the basis of literary data also
expresses the opinion on it that activation of dendrites at stimulation
of the surface of the cortex should proceed through the fibers of layer
I (Eccles, 1951). However, Dow even earlier proposed a similar
clarification to the spread of activity arising in the cerebellar
cortex during electrical stimulation of its surface (Dow, 1949).

At intensification of the electrical stimulation of the cortical
surface increase of amplitude occurs of the negative potential being
registered. This must be explained by the fact that at increasing
the intensity of the stimulation the number of fibers excited in
layer I increases. As a result of this, in the area of discharge
the number of synaptic terminals excited, under which excitation
in the dendrites arises, is increased. The amplitude of the dendritic
potential is increased in connection with intensification of stimulation
up to a certain limit, nevertheless; growth of amplitude of the
potential is limited by the number of synaptic terminals of the fibers
of layer I to the dendrites of the discharge portion.

Clarification, according to which activation of the dendrites is

realized through the fibers of layer I, ran into difficulty, which

consisted of this, that the quick (potential) which itself expresses

the current of effect of the excited fibers of layer I (see recordings

of the dendritic potentials in all the works published and in the

above-mentioned oscillograms) does not precede the slow dendritic

potential. Still in a number of cases it proved possible to register

the effects of direct stimulation of the surface of the cortex in

which the quick potential of complex character, which consisted of

a group of asynchronous impulses of axon origin (Fig. 17), preceded

the slow negative potential. This initial component is altered in

connection with the change of direction of the stimulating current

and in connection with change of place of stimulation, i.e. at

change of the conditions of stimulation of the surface of the cortex.

At prolonged stimulation at a rhythm of 50 per second many components

of this initial effect fall out.

The fact that quick potentials were not usually registered

before the slow is probably explained by the poor conditions for

discharging quick fluctuations from the surface of the cortex.

At the moving/the discharge electrode away from the point
of

stimulated the amplitude of the dendritic potentials being registered

is reduced, i.e. there is observed, as it were, a logarithmic decrement

spread of the activity. Eccles explains this phenomenon by the manner

of spread of fibers of layer I. This must be comprehended in the
following way: for example, excitation from the part stimulated
comes to point A through 10 fibers of layer I. From collaterals of
these fibers the dendrites placed here are activated, part of these
fibers terminating then and there. Impulses are admitted to point
B through 6 fibers, to point C through 3, etc. The degree of
decrement of the dendritic potential is proportional to the decrease
of the fibers excited.

As we have seen, not only the rate of spread, but also the
degree of decrement and, consequently, the distance of spread depend
on the depth of narcosis. During deep narcosis dendritic negative
potentials are registered in cat at a distance of 5 mm. from the
site of stimulation. In Burns' experiments (1951) on isolated
strip of non-narcotized cortex it was registered at a distance of
10 mm.; in the recordings presented as obtained on slightly narcotized
animal it was at a distance of 11 mm.

We have seen that during deep narcosis and generally at decline
of the functional state of the cortex first the activity begins to
spread to an even lesser distance and, secondly, its rate of spread
is reduced. This is perhaps connected with the effect of the narcotic
on elements responsible for the spread, i.e. the fibers of layer I.
It can be thought that these fibers are very sensitive to the action

of the narcotics and fall into a parabiotic state. Furthermore, it
is possible to assume that the stimulation applied to them at this
time causes excitation which spreads with decrement along the fibers
and their collaterals; the attenuated biocurrents of the terminals
closest to the collaterals are still capable of showing a stimulating
action on the neuronic elements and of stipulating the arising of
local potentials.

Thus, electrophysiological and histological data permit
concluding that electrical stimulation applied to the surface of
the cortex leads primarily to excitation of the fibers of layer I.
The excitation, spreading along the fibers and their collaterals,
reaches the synaptic endings that are located mainly on branches
of the top dendrites of the pyramidal neurons; the dendrites are
activated and generate bioelectrical potentials which are discharged
from the surface of the cortex close to the point stimulated, in
the form of negative potentials.

(Legend to Fig. 17, textpage 48: Quick potentials before
slow potentials. Cat No. 32, July 10. 1950. The discharge electrode
is placed on the surface of the gyr. sigmoideus. A - effects
registered when a point of the same convolution was stimulated at
a distance of 10 mm. from the discharge electrode. B - effects
registered when a point of the same convolution was stimulated at a

distance of 8 mm. from the discharge electrode (intensity of

stimulation in both cases 30 v.).)

Chang proposes an entirely different explanation for the arising

and spread of the potentials being considered. The slow negative

potential (discharged in his experiments at a distance of up to 5 mm.

from the stimulating electrodes) expresses the dendritic potential

itself, provoked by direct stimulation of the horizontal branches

of the top dendrites. The negative potential is registered when

the excitation impulses arrive through the branches of the dendrites

from the site of their stimulation under the discharge electrode.

The spread rate of these potentials corresponds to the excitation

spread rate in the dendrites (Chang, 1951).

However, as said, the horizontal branches of the top dendrites

have a length no greater than 2 mm.; in the experiments of Chang

himself the potential was detected at a distance of 5 mm. from the

site of stimulation and in the case presented in Fig. 11, D and E,

the potentials were registered at a distance of 11 mm., a fact which

it is by no means possible to explain from Chang's comment.

The position that the dendritic potentials arise at stimulation

of the surface of the cortex, provoked by direct stimulation of the

dendrites, Chang supports by the following facts: 1) In connection

with intensification of the stimulation of the cortex the amplitude

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of the dendritic potential relatively quickly reaches a maximal

magnitude. Chang thinks that if its origin was stipulated by

transmission of the excitation to the dendritic synapses, then

the amplitude of the potential would be increased within immeasurably

greater limits. 2) Dendritic potentials are not appreciably changed

under the effect of strychnine. This fact cannot, however, serve as

proof of the fact that dendritic potentials arise during stimulation

of the cortical surface without the agency of the synapses. It is

well known that strychnine does not appreciably change the amplitude

and character of local potentials of the motoneurons (arising through

double neuronic arcs, i.e. directly under the influence of afferent

impulses) as well as of local potentials arising in the elements of

layer IV of the cortex under the effect of afferent impulses (see

Chapter IV). Under the influence of strychnine a certain increase

occurs of the amplitude of these local potentials, but we see the

same too in respect to dendritic potentials provoked by stimulation

of the surface of the cortex. The spasmodic strychnine effect is

always connected with excitation of the intermediate neurons, which

the strychnine acts on selectively in the sense of elevation of

excitability.

It has already been said that the negative potential is registered

from the surface of the cortex and during point stimulation of the middle

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and deep layers of the cortex, the negative potential being registered
at a distance of several millimeters from the stimulating microelectrode
sunk into the cortex. In the opinion of Burns and Grafstein (1952)
this fact shows that the negative potential arises without participation
of the fibers of layer I, that it is stipulated by excitation of the
horizontal branches of the top dendrites, i.e. they think that during
stimulation in the depths of the cortex the top dendrites are excited
and then the excitation spreads along their horizontal branches in
layer I of the cortex. However, this fact is easily explained otherwise
if it is taken into consideration that the fibers of layer I arise
from the subjacent neurons, chiefly of the middle and deep layers of
the cortex. It is not remarkable that at stimulation of these elements
negative potentials arise: excitation spreads along the ascending
axons (or collaterals), then along their horizontal branches in layer I
and stipulates the arising of local potentials in the top dendrites
on which the fibers of layer I terminate synaptically.

        With the method of stimulation used and with narcosis stimulation
of the motor area of the cortex did not lead to the arising of movement,
whereas from the surface of the motor area of the cortex around the part
stimulated slow negative potentials of greater amplitude were registered.
Thus, in the dendrites of the pyramidal neurons excitation arose. The
pyramidal neurons as a whole were not excited. No discharges of
excitation into their axons occurred, i.e. into the pyramidal courses.

It is characteristic that when a negative potential is discharged
from the surface of the cortex in response to stimulation of the cortex,
then in the same part of the cortex, from the microelectrode found at
the level of the cellular bodies of the larger pyramidal neurons (at
a depth of 1.3 mm.) quick discharges are never registered (Burns and
Grafstein, 1952).

It is known that if by thermocoagulation the upper three layers
in field 4 die, then at stimulation of this part the same motor
reactions are provoked as prior to destruction by layers; the threshold
of electrical stimulation is not thereby altered. After destruction
of all layers of the cortex, only very powerful stimulations provoke
a motor reaction because of direct stimulation of the white matter
(Dusser de Barenne, 1933a, 1934). Since the threshold of provocation
of the motor reaction was not changed after thermocoagulation, then,
consequently, during the given conditions of experiment reactions were
provoked because of direct stimulation of the cellular elements of the
deep layers. It is also known that the threshold of provocation of·
motor reactions when there is stimulation of the motor cortex depends
on the method of stimulation (Dusser de Barenne, 1934a): at unipolar
stimulation it is lower than at bipolar stimulation, at which electrical
lines run mainly through the dendrites of the pyramidal cells. Hence,
it is possible to conclude that isolated stimulation of the dendrites
does not lead to excitation of the corresponding cellular bodies.

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On the basis of the data of Clare and Bishop (1954) it likewise
is possible to conclude that when the top dendrites of the association
pyramidal neurons undergo direct electrical stimulation (stimulating
microelectrodes at a depth of 0-0.3 mm.), then discharge of excitation
impulses into their axons does not occur; when the (cellular) bodies
of the association pyramids (the microelectrodes were sunk into layer
IV) are stimulated directly, then discharges of impulses in their
axons arise under the effect of the same stimulation.

The facts obtained in experiments with simultaneous registration
of biopotentials from different layers testify that when excitation
arises in the dendrites under the effect of impulses from the fibers
of layer I, the (cellular) bodies of the pyramidal neurons are not
aroused by impulses into the axons; likewise, no local excitation arises
in them that would be expressed in registration of a characteristic
negative potential from elements of the deep layers. Consequently,
excitation of dendrites of the pyramidal neuron does not lead to
excitation, spreading or regional, of the remaining parts of the neuron
(cellular body and axon). Thus, when excitation impulses come to the
top dendrites of the pyramidal neurons, the reaction is limited by
the arising of regional excitation of the top dendrites.

With weak single stimulations of the cortex and with deep
narcosis and strong stimulations a simple bioelectrical reaction
arises, a negative biopotential, the minimal length of which is

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equal to 10 milliseconds. The minimal length of such a negative

potential developed by the motoneurons and by the intermediate

neurons of the spinal cord of cat is the same. On the basis of

this it is possible to assume that the length of the elementary

local potential, i.e. of the regional excitation, is approximately

identical for the various neurons of the central nervous system,

for instance for the neurons of the spinal cord and of the cerebral

cortex. (Footnote: At direct electrical stimulation of the optic

covering of frog the negative potentials discharged from its surface

likewise have a length of about 10 milliseconds (Roitbak, 1952).)

On the basis of experiments with simultaneous discharge of

biopotentials from the various layers of the cortex it was concluded

that the level at which alteration of the sign of the potential

occurs lies somewhere close to the surface, apparently at the

boundary of layers II and III: a negative potential expressing

regional excitation of the dendrites is registered only from layers

I-II (Fig. 16). At deeper placement of the discharge electrode in

the cortex, they stop discharging any considerable effects or they

change their sign. Hence, it is possible to conclude that regional

excitation (and a local potential corresponding to it) arising in

the top dendrite does not spread downwards through the dendrite for

any considerable distance and that it is virtually limited to the

place of its arising under the excited synaptic endings. If we

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were persuaded earlier of this, that bioelectrical potentials are

extremely local in respect to spreading along the surface of the

cortex and that the loops of stimulating current with the stimulation

method in question are not spread through the cortex to such a degree

as to cause excitation of the nerve elements at a point of the cortex

laying in line with it, then too it is possible to conclude in regard

to the physical spread of the biocurrents and of the stimulating

currents in the depths of the cortex: this issues from the possibility

indicated of isolated activation of surface layers of cortex. However,

finally, in natural conditions of activation of the cortex isolated

activation of the surface layers cannot proceed. As we have seen,

the main source of the fibers of layer I consists of the ascending

axons of cells with short axon and the recurrent collaterals of the

pyramids. Thus, the excitation of the fibers of layer I and the subsequent

activation of the top dendrites (and other elements of the surface layers)

presumes the preliminary excitation of the neurons of other layers of

the cortex, particularly of the neurons of layer IV, which is the main

regional ending of the afferent fibers of the cortex.

Thus, on the basis of an analysis of the dendritic potentials

of the pyramidal neurons it is possible to make the following two

basic theoretical conclusions: 1) excitation impulses arriving at

the dendrites provoke in them regional nonspreading excitation; 2)

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regional excitation of the top dendrites does not lead to excitation

of the corresponding pyramidal neurons. (Footnote: This conclusion

was made on the basis of the results of pointed experiments with

exposure of the cerebral cortex. Hence, as A.B. Kogan pointed out,

the following objection is possible: during exposure of the cortex

the top dendrites fall into a parabiotic state and, in response to

the impulses that come to them, respond with regional excitation.

Normally they conduct excitation to the body of the cell.) These

conclusions agree with the conclusions of Beritov concerning the

activity of the dendrites, that were made on the basis of an

analysis of numerous facts from the histology and physiology of

the central nervous system (see Beritov, 1941, 1948, 1949, 1953).

They do not agree with the prevalent concept, according to which

the dendrites conduct excitation to the body of the cell and even

are detectors "collecting" the stream of nerve impulses from different

sources and transmitting them to the axon (Gesell, 1940; Poliakov,

1953).

In virtue of the fact that the pyramidal neurons were oriented

vertical to the surface of the cortex, experiments with deep sinking

of the discharge microelectrode from the surface of the cortex vertical

to the white matter permit concluding on the profile of the outer

electrical field, which arises at regional excitation of the dendrites:

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points around the part excited, i.e. around the top dendrite, are

negative, and the points around the cell body and axon are positive

in respect to the remote "indifferent" point (Fig. 18, textpage 52:

Potentials opposite in sign, recorded from different parts of the

pyramidal neuron at excitation of its top dendrite. P - stimulating

electrodes on the surface of the cortex. $0-0_1$ - conditional level

at which the "inversion" of potential occurs. The scheme was con-

structed on the basis of results of electrophysiological experiments.)

Beritov thinks that dendrites do not conduct excitations

because the fine, bare bifurcations of the dendrites develop an

active process of little intensity, and this process up to the body

of the cell is not in a condition to spread, owing to the biocurrent

that thereby arises (Beritov, 1948, 1949). However, it can be thought,

in addition, that the neuroplasm of the dendrites is different in its

properties from the neuroplasm of the cellular bodies. For instance,

the various staining properties of the cellular bodies and of the

dendrites to be detected even with Nissl's method indicate this.

Finally the fact that excitation of the dendrites during natural

conditions of their stimulation (i.e. under the effect of the excitation

impulses conducted to them by the fibers) does not lead to excitation

of the neuron is perhaps explained likewise by the fact that the

presynaptic fibers terminate differently on the bodies of the neurons

and their dendrites (see below).

Sherrington concluded that the axon type of excitation
conduction is natural not only to axons of nerve cells but also
to their bodies and their dendritic offshoots and that the synapse
by its unique structure is capable of changing this type of
excitation conduction. In regard to the dendrites, this conclusion
was made by him on the basis that the dendrites may be nerve fibers
having cerebral-spinal ganglia in the form of a neuron (Sherrington,
1906). However, as Malone rightly notes (1932), it is necessary
to distinguish relationships in the central nervous system and in
the posterior-root ganglia and it is impossible even to assume that
dendrites of the central nervous system are similar in their properties
to sensory-nerve fibers.

Up to the present time the idea that dendrites of all neurons
conduct excitation in a way similar to that by which the peripheral
nerve fiber conducts it was prevalent, but facts obtained by oscillographic
study of the central nervous system have led recently to a change of
opinions on this statement by a number of American physiologists.

Chang thinks that at electrical stimulation of the top dendrites
excitation spreads not only through their horizontal branches, but
also through the dendritic trunk downwards to the body of the pyramidal
neuron at the rate of 1-2 m. per second. Excitation of the dendrites,
according to Chang, is normally transmitted to the body of the pyramid

and then to the axon. However, during narcosis the excitation of the

dendrites does not provoke excitation and discharge of the corresponding

pyramidal cell, since blockade occurs of the excitation at the site of

the transition of the body of the cell into an axon. By this is explained

the fact that the shock of electrical stimulation applied to the surface

of the motor area of the cortex, provoking a negative potential, does

not provoke a motor reaction. Nevertheless, as was pointed out, the

local potential is registered only at the level of layers I and II.

Thus, there are no bases for considering that the excitation reaches

the body of the pyramidal neuron and is blocked at the place of

emergence of the axon.

The difference in methods of activation of the pyramidal

neuron, through the cell body or through the dendrites, according

to Chang, consists of this, that because of the low rate of excitation

conduction through the dendrites the latent period of excitation is

in the second case considerably (by 3.5 milliseconds in his estimation)

greater (Chang, 1951).

However, later on and issuing from histological data on the

presence of two types of synaptic connections between the cortical

neurons, the axosomatic and the axodendritic, Chang came to several

other views. In his opinion, in connection with the fact that

presynaptic fibers terminate on the body of the pyramidal neuron

with numerous thickly distributed synapses, excitation of these fibers

usually causes the discharge of the pyramidal neuron in question;

presynaptic fibers on the dendrites of a pyramidal neuron terminate

with a moderate number of linearly distributed synapses, and excitation

of these fibers usually does not lead to the discharge of the pyramidal

neuron in question; the discharge can proceed with simultaneous

excitation of a great number of paradendritic synapses (Chang, 1952).

At excitation of the body of the pyramidal neuron the excitation

spreads, according to Chang, on the one hand, into an axon, and, on

the other, upward through the dendrites to the surface of the cortex

(Chang and Kaada, 1950). This universally adopted point of view,

according to which the dendrites conduct the excitation, is also held

by Lorente de No. On the basis of a study of the biopotentials of

neurons of the nucleus of a sublingual nerve at their antidromic

excitation, he came to the conclusion that the rate of spread of

excitation in the nerve cell (body of the cell + dendrites) is of

the order of 2 m. per second. He thinks that the antidromic impulses

spread along the dendritic bifurcations by the same principle as in

the nerve fiber, i.e. because the current of excitation subsequently

stimulates portions of dendrites along the travel line of the excitation.

Likewise, he considers the question unsolved whether impulses reach

the very fine bifurcations of the dendrites (Lorente de No, 1947).

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Bishop and Clare (1953) on the basis of the data obtained in experiments with discharge of potentials from different layers of the cortex at stimulation of its surface formed the conclusion that dendrites of pyramidal neurons do not conduct excitations downward to the body of the cell, i.e. entirely the same conclusion as was made by me on the basis of like experiments. However, on the other hand, on the basis of experiments with stimulation of the deep layers of the cortex, they reached the conclusion that dendrites of pyramidal neurons conduct excitation from the body of the cell upward to the surface of the cortex. We still come back to paradoxical conclusions, according to which dendrites do not conduct excitation orthodromically (i.e. to the body of the cell), but antidromically (i.e. from the body of the cell).

(Legend to Fig. 19, textpage 54: Oppression of "spontaneous" electrical activity during stimulation of the surface of the cortex. Recordings A and B - cat No. 26, June 3, 1950. Relatively deep nembutal sleep. The stimulating electrodes and discharge electrode are placed on the surface of the gyr. suprasylvius. The discharge electrode is a distance of 2.5 mm. from the stimulating electrodes. A - beginning of tetanic stimulation (frequency 50 per second, intensity 30 v.). B - after 10 seconds of tetanization; end of stimulation.

Recordings C and D - cat No. 24, May 20, 1950. Relatively
deep nembutal sleep. The stimulating electrodes (P) are placed on
the gyr. sigmoideus post.; potentials are discharged simultaneously
from a point on the surface of the same convolution at a distance
of 2 mm. from P (E_1, upper curves) and from the surface of gyr.
suprasylvius 10 mm. distant from P (E_2, lower curves). C - beginning
of short tetanic stimulation (50 per second, 25 v.). D - end of
stimulation. E - scheme of arrangement of electrodes (Roitbak,
1953a).)

Inhibition of "spontaneous" electrical activity. In the
course of investigation we were repeatedly confronted with facts
evidently related to the phenomenon of cortical inhibition.

At the time of stimulation of the surface of the cortex at
a rhythm of 50-100 per second, attenuation and even complete oppression
of "spontaneous" electrical activity (Fig. 19) can occur; reduction of
it occurs after 0.5-1 second through cessation of stimulation.
Oppression of "spontaneous" electrical activity proceeds at those
points of the cortex at which negative slow fluctuations occur in
response to stimulation, i.e. during deep narcosis this occurs in
a small territory around the stimulating electrodes. At those points
of the cortex where the stimulation in question does not cause
negative biopotentials "spontaneous" electrical activity is not
altered (Fig. 19, C and D). Oppression of "spontaneous" electrical

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activity at tentanization of the surface of the cortex occurs too
when, as a consequence of local strychninization, this activity is
sharply intensified (Fig. 20, textpage 55: Oppression of "spontaneous"
electrical activity at stimulation of the surface of the cortex. Cat
No. 12, Dec. 2, 1949. Stimulating electrodes and discharge electrode
on the surface of the gyr. suprasylvius; distance between them 2 mm.
11 minutes after local poisoning of the brain under the discharge
electrode with a 0.1% solution of strychnine. A - intensification of
electrical activity (after poisoning) and beginning of tetanic
stimulation (50 per second, 25 v.). B - immediate continuation of
recording A. C - electrical activity 0.5 second after cessation of
stimulation. For this period of 0.5 sec. the electrical activity
remained depressed.).

On the basis of the facts cited it is possible to assume that
oppression of "spontaneous" electrical activity during tetanization
of the surface of the cortex is causally connected with the negative
slow potentials arising thereby, i.e. that oppression of "spontaneous"
electrical activity is stipulated by excitation of the dendrites in the
surface layers of the cortex.

It would be possible to give this phenomenon another explanation
by admitting that the "spontaneous" activity is stipulated mainly by
the activity of the nerve elements of the surface layers of the cortex

and that the elements activated as a result of the tetanizing

stimulation no longer produce rhythmical fluctuations of "spontaneous"

activity. However, it is known that after thermocoagulation of the

upper two layers of the cortex "spontaneous" electrical activity of

the part in question does not disappear but only attenuates somewhat

(Dusser de Barenne and MacCulloch, 1935, 1936). Recently it was

established that during comparatively weak stimulations of the surface

of the cortex at 50-100 a second a prolonged negative potential of

a nonfluctuating character arises (Beritov and Roitbak, 1953). The

amplitude of this potential is increased at increase of frequency of

the stimulation and reaches 1 milliv. and more. The slow negative

potential reaches a maximal amplitude 1-1.5 seconds after the beginning

of tetanization of the surface of the cortex and lasts 3-4 seconds,

gradually attenuating, (Fig. 21, textpage 56: Long potentials of

nonfluctuating character that arise during electrical stimulation of

the surface of the cortex. Cat under deep nembutal narcosis. On the

surface of the gyr. suprasylvius the stimulating electrodes were placed

and 3 and 10 mm. distant from the discharge electrodes. Booster with

a greater time constant. Length of irritating stimuli 0.5 millisecond.

A - frequency of stimulation 9 per second. B - frequency of stimulation

50 per second. C - effect of repeated stimulation after 10 seconds.

Intensity of stimulation in experiments A - C 10 v. D - intensity of

stimulation 5 v., frequency 100 per second. Deflection upward

denotes negative character of discharge electrode very close to

the point of stimulation (Beritov and Roitbak, 1953).).

With the beginning of the development of the negative potential

the "spontaneous" electrical activity immediately attenuates or ceases

and remains oppressed throughout all the time of the beginning of

this potential. Thus, regional, fixed, nonfluctuating excitation

of the system of dendrites in the surface layers of the cortex

stipulates inhibition of activity of the neuronic elements of the

cortex.

On the other hand, during comparatively intense stimulations

of the surface of the cortex at a rhythm of 50-100 per second a

prolonged positive potential can arise that is attended by intensification

of the "spontaneous" electrical activity (Beritov and Roitbak, 1953).

When from the surface of the cortex a long negative biopotential

is discharged, the inner layers of the cortex are polarized in a positive

way (Fig. 15). It can be thought that activation of the dendritic

plexus of layers I and II stipulates the anelectrotonization of the

cellular bodies of the pyramidal neurons in the inner layers of the

cortex and by this very thing oppression of "spontaneous" electrical

activity.

The facts obtained at passage of a constant current through the

cortex can serve as argument for the correctness of this assumption

(Burns, 1954). If to a small part of the surface of the cortex an

anode was applied, then with a current energy of about 100 microamperes

excitation occurred of the cortical neurons. This phenomenon did not

arise with a reverse direction of the current. In another series of

experiments a microelectrode was introduced into the cortex to a depth

of 1.2 mm., i.e. into layers V-VI. When it was connected with a

cathode of constant current, then an excitation arose of the cortical

neurons. When it was connected with the anode, no excitation arose.

Intense rhythmical stimulation of isolated non-narcotized strip

of cortex aroused excitation of neurons of the deep layers of the part

stimulated, with prolonged aftereffect. If at this time to the surface

of this part of the cortex a cathode of constant current was applied,

then the aftereffect broke immediately. The same effect was obtained

if the anode was applied to the microelectrode introduced into layers

V-VI of this part of the cortex. Thus, during negative polarization

of the summit dendrites inhibition occurs of the activity of the

pyramidal neurons of a given point of the cortex as a consequence of

positive polarization of their cellular bodies: the catelectrotonus

of the dendrites is associated with the anelectrotonus of the cellular

bodies. Is it impossible to compare this phenomenon with pericatelectro-

tonus, with the phenomenon of anodic reduction of the excitability in

the region of the neuron adjacent to the region of the catelectrotonus?

On the basis of the facts just cited it is possible to conclude that during artificial polarization of the cortex when the cathode is on its surface there is created an electrical field the same in configuration as in the case of natural excitation of the top dendrites through tetanization of the fibers of layer I that bring about their excitation.

Vorontsov (1949) thinks that at excitation of the dendritic ramifications very weak currents should arise that may show only an insignificant effect on the bodies of neurons. However, he considered the results of excitations of the very fine ramifications of the dendrites. We too have seen that during regional excitation of the top dendrites of the pyramidal neurons a positive potential of great amplitude is registered from the bodies of these neurons.

In connection with what has been said above, the following fact which we have already mentioned is of interest. In experiments with registration of biopotentials of the individual motoneuron through a microelectrode inserted therein it has been established that when exciting afferent impulses reach the motoneuron, then an ordinary local potential (negative) arises in it, which we have already spoken of above. When inhibiting afferent impulses arrive at the motoneuron, then a positive potential of the same length and form as the negative local potential is registered from the motoneuron

(Eccles, 1952, 1953). Thus, the process of regional excitation and

the process of inhibition have an electrical display opposite in sign.

Proceeding from the dendritic hypothesis of inhibition (Beritashvili,

1953), it is necessary to assume that inhibited impulses are impulses

coming to the synaptic endings on dendrites of the motoneuron. The

body of it is thereby polarized in a positive way, as we saw in regard

to the body of the pyramidal neuron during regional excitation of its

dendrites (see Roitbak, 1955, for more detail).

Contribution to the question of the lability of the dendrites.

As known, the higher boundary rhythm of excitation is the standard

of lability (functional liveliness). With characteristics of lability

these bring a reaction which is most specific to the tissue in question

(Ukhtomskii, 1939-40). The lability of the nerves is determined by

the higher rhythm of the currents of effect, since the excitation

that is spread is most specific to their reactions. As we have seen,

regional excitation for the dendrites is a specific reaction, and if

the same principle is applied here, then the lability should be

determined by the higher rhythm of the local potentials.

The higher rhythm so determined for the dendrites is equal at

deep narcosis to 100-125 per second. The lability quickly drops in

the course of stimulation: the rhythm of stimulation (50-100 per

second) is reproduced only at the very beginning of stimulation, then

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the effects quickly attenuate and go down to nothing (Fig. 12).

Thus, stimulation frequencies of 50-100 per second for the cortical

surface can, as it were, be noted as the worst; the optimal will be

frequencies of 10-20 per second.

In Vvedenskii's experiments with nerve-muscle preparation

(1886) the worst state was summed up in the nerve-muscle lamina.

In experiments on it with strychninized spinal cord (1904) it is

in the motor neurons of the anterior horn. In the oscillographic

experiments of Beritov and Roitbak (1950a) on strychninized spinal

cord it is in the intermediate neurons. But when this state is

summed up during stimulation of the surface of the cortex, in the

fibers of layer I, in their synaptic endings, or in the dendrites,

on what principally are these terminations found? The nerve fibers,

the conductors, always possess greater lability than their station

of destination. This evidently is the general rule and the fibers

of layer I of the cortex, in spite of the fact that the greater part

of them are devoid of myelin, hardly present an exception. However,

there are also factual indications that during prolonged several-

second stimulations of the surface of the cortex at a rhythm of 50-

100 per second the nerve fibers of layer I continue conducting

excitation and transmit it through the synapses to the neuronic

elements (see Chapter III).

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Thus, it should be concluded that the phenomena of intensification
and of attenuation of the bioelectrical potentials being considered during
changes of frequency of stimulation of the cortical surface reflect the
very processes occurring in the dendritic offshoots of the pyramidal
neurons.

Certain preceding discussions lose their significance if the
original position is inaccurate, that the higher rhythm of the local
potentials is the criterion of lability for the dendrites. Indeed
can the rhythm of local potentials testify unreservedly to the
functional liveliness of the dendrites or of the cellular bodies?
As said, at application to the spinal sensory nerve of subthreshold
(in the sense of provocation of the reflex discharge in the motor
nerve) shock of stimulation in the corresponding motoneurons of the
spinal cord a regional excitation or local potential develops and
after 10 milliseconds attenuates. If stimulations are applied with
such calculation that from each subsequent shock of stimulation
afferent impulses come to the motoneurons even to attaining the
preceding local potential in its height, then the phenomenon is
observed of summation of local potentials: a nonfluctuating slow
potential of greater amplitude arises, i.e. the curve of the bioelectrical
potential no longer reflects the rhythm of the stimulation. However,
it is absurd to consider this the expression of the worst state of
the motoneurons, since just the phenomenon of summation of regional
stimulation lies at the basis of the excitation, of the discharge of

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the neurons. The same refers also to the local potentials of the
nerve fibers. Dendritic potentials cease to arise through the
rhythm of stimulation at irritability frequencies of 50-100 per second,
but it is incorrect to consider these frequencies as the worst. A
nonfluctuating potential thereby arises, the amplitude of which is
higher than the amplitude of the elementary dendritic potentials
that arise at individual shocks of stimulation. Its amplitude is
increased at increase of frequency of stimulation within the limits
of 20-100 per second. This nonfluctuating dendritic potential is
not an epiphenomenon, but is connected, as we see, with a process
of inhibition.

Certain high frequencies of stimulation at which a nonfluctuating
potential arises less in intensity and length than during certain
lower frequencies can perhaps be considered as the "worst" frequencies
in regard to local potentials. For instance, in the experiments of
Delov and Lapitskii (1935) the slow potential discharged from the
surface of the spinal cord of frog during stimulation of the sciatic
nerve at a stimulation frequency of 100 per second reached an amplitude
of 1 milliv. and lasted for a long time during uninterrupted stimulation.
At a stimulation frequency of 200 per second the potential after a
small rise (0.3 milliv.) quickly dwindled to nothing. Or, for instance,
the slow nonfluctuating potential discharged from the surface of the

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medulla oblongata of frog during stimulation of the sciatic nerve

has at a stimulation frequency of 100 per second a lesser amplitude

than at a frequency of 10 per second; at a frequency of 100 per

second during uninterrupted stimulation it quickly attenuates,

whereas at a frequency of 10 per second it preserves for a long

time its initial amplitude (Roitbak, 1952).

2. Supplementary Negative Potentials

During deep narcosis the bioelectrical potentials that arise

in response to direct electrical stimulation of the surface of the

cortex are relatively simple and constant. They are altered from

intensity, frequency, and duration of stimulation in a certain

simple dependency on these factors, which was pointed out in the

first part of this chapter. However, at light narcosis in a way

like that by which motor reactions on the terminations are complicated

and become altered (but not stereotypic as they are at deep narcosis)

from direct electrical stimulation of the motor area of the cortex

(Ukhtomskii, 1911), the bioelectrical responses of the cortex are

complicated and become altered at direct electrical stimulation of it.

These complex bioelectrical effects will be examined further on.

Further back a detailed analysis was given of those potentials

simple in character which express regional excitation of the top

dendrites of the pyramids that sets in immediately under the effect

of impulses from the fibers of layer I. The characteristic bioelectrical
reaction to stimulation of the cortical surface during deep narcosis
is a negative biopotential of 10 milliseconds' duration. For instance,
in recordings A and B of Fig. 12 the first shock of stimulation causes
this simple bioelectrical reaction, a negative potential lasting 10
milliseconds and reaching an amplitude of 0.75 milliv. Subsequent
shocks with a stimulation frequency of 3 per second (recording A) and
16 per second (recording B) cause potentials of greater amplitude
(1.2 milliv.) and of greater duration (up to 40 milliseconds).

The increase of amplitude and duration of the negative slow
potentials occurs because of the rise of additional negative fluctuations.
In Fig. 8 are indicated the first 2 effects of stimulation at a rhythm
of 16 per second. Additional fluctuations are indicated by arrows.
The first shock of stimulation causes an elementary dendritic potential
not complicated by supplementary fluctuations. The second shock provokes
a more complex effect: the elementary negative potential is complicated
by two additional negative fluctuations. They arise on the descending
part of the potential and stipulate increase of the length of the effect.
At a stimulation frequency of 1 per second and less the effects remain
simple, and in response to single shocks of stimulation stereotypic
responses arise.

Supplementary fluctuations can be expressed not only in the
form of "humps" on the background of the main dendritic potential but

can be fluctuations of considerable amplitude sharply separate from
the first negative potential. During certain conditions their
amplitude can considerably exceed the amplitude of the initial negative
potential.

(Legend to Fig. 22, textpage 60: Supplementary negative
potentials. A, B, and C - cat No. 23, May 17, 1950. Stimulating
electrodes and discharge electrode on the gyr. suprasylvius; 2.5
mm. between them. Stimulation frequency about 12 per second. A -
stimulation intensity of 5 v., B - 10 v., C - 30 v. D - cat No. 30,
July 1, 1950. Stimulating electrodes are placed at the anterior
pole of the gyr. suprasylvius; at a distance of 4 and 10 mm. from
them in the same convolution are placed the discharge electrodes E_1
and E_2. The potentials are registered simultaneously from point E_1
(lower curve) and E_2. Stimulation intensity 30 v., frequency 10 per
second. E - cat No. 9, July 28, 1949. Distance between stimulating
electrodes and discharge electrode 4 mm. Intensity of stimulation
16 v., frequency about 13 per second. F - after one minute of
stimulation, E_1 - effect of twice as powerful stimulation 30 seconds
after the heart has stopped. G - cat No. 32, July 10, 1950. Stimulating
electrodes and discharge electrode are placed on the gyr. sigmoideus
post. (P - E = 4 mm.). Stimulation intensity 30 v., frequency 10
per second. Stimulation did not provoke motor reaction of the animal.)

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These supplementary waves arise first in connection with
intensification of the stimulation (Fig. 22, A-C), secondly, as we
have already seen, in connection with the increase of frequency of
the stimulation, and finally in connection with repeated stimulations
(Fig. 22, D).

During stimulation at a rhythm of 10 per second these additional
fluctuations can be intensified through the course of the stimulation
(Fig. 22, D and G). In a number of cases in the course of the stimulation
the latent period of their arising is reduced. During prolonged
stimulations the supplementary fluctuations attenuate in a greater
measure than the initial negative potential. For instance, in one
experiment, the beginning of which is presented in Fig. 22, D, the
2d, 3d, 4th, and 5th, shocks of stimulation caused double effects,
but already the 6th shock and all subsequent ones ceased to provoke
a supplementary negative potential. Under the effect of strychnine
additional fluctuations are extremely intensified (Fig. 23), whereas
the initial negative potential increases very little (Footnote: A
certain intensification of the initial negative potential under the
effect of strychnine indicates that under the effect of strychnine
intensification occurs of local potentials, i.e. of regional excitation.
An analogous conclusion was made on the basis of experiments on the
spinal cord (Beritov and Roitbak, 1950b).) During strychninization
the latent period of a supplementary fluctuation is reduced (Fig. 24)

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and it can unite with the first, as the result of which a single

prolonged potential arises. During stimulation at a rhythm of 10

per second alternation of effects complicated by gigantic supplementary

fluctuations can be observed with simple effects (Fig. 23), i.e.

during repeated stimulations supplementary fluctuations, intensified

under the effect of the strychnine, can separate out (compare Chang,

1951a).

(Legends on textpage 62, Fig. 23: Effect of strychnine on

cortical bioelectrical effects caused by electrical stimulation of

the cortex. Cat No. 28, June 10, 1950. Nembutal. Stimulating

electrodes (P) - on the gyr. suprasylvius. The potentials are

discharged from the same convolution at a distance of 4 mm. from

P (E_1, upper curves) and from a point of the gyr. suprasylvius of

the opposite hemisphere, symmetrical to the point stimulated (E_2,

lower curves). Stimulation frequency 10 per second, intensity 30 v.

A - prior to poisoning, B and C - 5 minutes after local poisoning

with strychnine (1%) of point E_1. D - effect of stimulation at a

rhythm of 3 per second after poisoning of point E_1 with a saturated

solution of strychnine.

Fig. 24: Effect of strychnine on the biopotentials of the

cortex that were provoked by its electrical stimulation. Cat No. 12,

Dec. 2, 1949. Nembutal. The stimulating electrodes and discharge

electrode are placed on the gyrus suprasylvius; the discharge electrode

is found at a distance of 2 mm. from the stimulating electrodes. A -

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the effect of a single stimulation (25 v.) prior to poisoning. B -
10 minutes after local poisoning of the cortex under the discharge
electrode by 0.1% solution of strychnine. C - 15 minutes after
poisoning (in experiment C - intensification is reduced).)

During very light narcosis or in experiments on non-narcotized
preparations the very first shock of stimulation produces complex
effects with supplementary fluctuations (Fgi. 25, textpage 63:
Bioelectrical potentials registered on the surface of the cortex
near the point stimulated in non-narcotized cat. Cat No. 31, July
4, 1950. The larger hemispheres (the cerebrum) are revealed, the
spinal cord is intersected at the boundary of the medulla oblongata,
artificial respiration. P-E$_1$ distance = 3 mm., P-E$_2$ = 7 mm.
Stimulation intensity 30 v., frequency 10 per second. A is the
beginning and B the end of brief stimulation (shortly after this
recording the functional state of the cortex worsened greatly).).

The facts cited permit concluding that the supplementary
negative potentials are stipulated by the activity of the intermediate
cortical neurons. This indicates:

1) Their disappearance during a worsening of the functional
state of the cortex or their sensitivity to narcosis. As Chang
(1951) pointed out, during anoxia supplementary fluctuation disappears
after 1 minute; the initial negative potential, as already said,
disappears after 1.5 minutes.

2) Their quick attenuation during long exhaustive stimulations.

3) Their intensification in connection with intensification and increase in frequency of the stimulation.

4) Their senstivity to strychnine.

Histological information on layer II of the cortex. Axonal plexus of layer II. In layer II there is a complex plexus of axon terminations, in which the short axons of the cells of layer II, the collaterals of the fibers of layer I, and the ascending axons of the cells of layer III participate (Lorente de No, 1933).

Types of cells of layer II. 1) Star cells (modified pyramids). These are cells of average size with numerous dendritic offshoots proceeding in all directions and supplied with spines. The axon runs into the white matter, giving off 6-10 collaterals into layers I, II, III, V, and VI (Lorente de No, 1933; O'Leary and Bishop, 1938).

2) Cells with horizontal axon. They are distinguished from the preceding by their axon: it has a horizontal direction and gives off a great number of short collaterals that spread into layer II and terminate, according to Lorente de No, on the bodies of the star cells.

3) Cells with ascending axon. They are of a lesser size. Their dendrites spread into layers I and II, but the axon, at having attained layer I, divides into two tangential fibers, each of which gives off collaterals (Lorente de No, 1933).

4) Cells with dendrites proceeding with two tufts into layers

I and III. The axon or its branch proceeds into layer III, terminating

there around the pyramids (O'Leary and Bishop, 1938).

In Fig. 26 layer II is schematically presented, its neuronic

elements and their connections. (Legend to Fig. 26, textpage 64:

Schematic depiction of the neurons of layer II of the cortex and of

certain of their connections. 1, 2, and 3 are the main types of

neurons of layer II of the cortex. 1 is a neuron with ascending

axon. 2 is a neuron with horizontal axon. 3 is a neuron with

descending axon and collateral returning into layer I (star cell).

The scheme was composed on the basis of certain histological data.)

On the basis of certain histological information mentioned

above about layer II of the cortex it is possible to make the following

conclusions:

1. During excitation of the system of fibers of layer I

excitation of the cells of layer II can occur, because in layer II

there are numerous collaterals of the fibers of layer I.

2. During excitation of the cells of layer II activation can

occur of the top dendrites in layer I, because among the cells of

layer II are cells with axon ascending into layer I, which take on

there a horizontal direction and give forth many collaterals. In

addition, the axons of the star pyramids give forth collaterals into

layers I and II.

3. During excitation of the cells of layer II activation can occur of elements of the deep layers of the cortex, since the axons of the star pyramids give forth collaterals into layers III, V, and VI, and certain cells with short axon of layer II terminate in the pyramids of layer III.

4. During excitation of the horizontal cells activation should proceed of the entire complex of star pyramids and, thanks to the latter circumstance, intensified activation of the deep layers.

Origin of supplementary negative fluctuations. There is no doubt that the supplementary negative fluctuations in the effects that arise in response to stimulation of the surface of the cortex are stipulated by excitation of the intracortical neurons. Histological data give indication that they should be stipulated by the activity par excellence of the neurons of layer II of the cortex. The mechanism of their arising in the simplest case can be connected with excitation of the cells of layer II with axon ascending into layer I (Fig. 26 - 1).

During stimulation of the fibers of layer I impulses of excitation proceed first to the synaptic endings of the fibers of layer I for the top dendrites and, secondly, to the cells of layer II with ascending axon, the endings and collaterals of which form additional synaptic fields for the top dendrites. As a result,

immediately in response to the stimulation, the first negative

fluctuation arises, already designated by us the elementary

dendritic potential. In the cells of layer II there also arises

a regional excitation, and (when there is a good functional state

of the neurons), when it reaches a certain size, these neurons are

discharged by impulses into their ascending axons, which stipulates

additional "impulsation" to the top dendrites of layer I, the

arising in them under the synaptic endings of the ascending axons

of a regional excitation, and the arising of a supplementary nega-

tive fluctuation in the effects being registered.

During participation of cells with short axon that branch

for a short distance in the limits of a given layer (typical Golgi

II cells), cells which are extremely numerous in all layers of

the cortex, an association and involvement in the reaction of the

new complexes of cells with ascending axon can occur, which readily

explains the fact of the arising of a series of supplementary

fluctuations.

Excitation of the neurons of layer II proceeds as a result

of the spread (in connection with intensification of stimulation)

or of the temporary (in connection with increase of frequency of

stimulation) summation of excitation. Frequency of 10 per second

is evidently an optimal frequency of stimulation for excitation of

these neurons governing the arising of additional negative potentials.

During strychnine poisoning, in consequence of the elevation of excitability, a greater number of intracortical neurons are drawn into reaction and their activity is synchronized. A result of this is that for the top dendrites of the pyramids there sets in at each stimulation an incomparably greater number of impulses from the intermediate neurons. Because of this, intensification of a secondary negative fluctuation and the arising sometimes of a whole series of additional fluctuations occur.

At stimulation of the surface of the cortex (during excitation of the fibers of layer I) not only neuronic elements of layers I and II can come into an active state but also the neuronic elements of the deeper-lying layers as a result of the excitation, for instance, of the star pyramids of layer II (Fig. 26 - 3). This should lead to the arising of new bioelectrical phenomena, to a consideration of which we shall proceed immediately.

3. Positive Potentials

At placement of the discharge electrode more deeply into the middle and deep layers of the cortex the sign of the potential provoked by stimulation of the surface of the cortex changes. To this phenomenon was given an explanation, according to which the

arising in the dendrites of the pyramidal neuron of a local potential
and, consequently, of a difference of potentials between the dendrites
and the other parts of a given neuron leads to positive polarization
of the latter, i.e. of the cell body and axon. It is possible to
think that in case of the arising of a regional excitation in the
bodies of the pyramidal neurons a positive potential will be registered
from the surface of the cortex (from their top dendrites).

(Legend to Fig. 27, textpage 66: The arising of positive
potentials in connection with increase of frequency of stimulation
of the surface of the cortex. Cat No. 27, June 7, 1950. Gyrus
suprasylvius. Distance between discharge electrode and stimulating
electrodes 4 mm. Intensity of stimulation 30 v. A - frequency of
stimulation 15 per second. B - frequency of stimulation 40 per
second.)

Adrian in his works with stimulation of the surface of the
cortex and registration of the bioelectrical reactions arising
established that during a good functional state of the cortex shocks
of stimulation, for instance at a rhythm of 10 per second, provoke
negative potentials at first, then the sign of the potentials changes,
i.e. positive fluctuations begin to arise, the amplitude of which
grows in the course of the stimulation, the potentials may become
more complex in connection with the arising of additional positive
fluctuations (Adrian, 1936).

This phenomenon was observed too in my experiments. Conversion of negative potentials to positive in the course of the stimulation occurs the more quickly the more frequent the stimulation up to a certain limit. At a stimulation frequency of 10 per second this can proceed only after several seconds of stimulation, as this was in one of the experiments of Adrian. At a frequency of 40 per second this can proceed after 0.2 sec. (Fig. 27), but in one experiment this proceeded after three shocks of stimulation (Fig. 36, C). On non-narcotized rabbit it is possible to observe how even at a frequency of stimulation of 3 per second the effects after several seconds of stimulation change their sign. On narcotized cats this change usually occurs only at a certain relatively greater frequency of stimulation (20-50 per second). At lesser frequencies of stimulation this phenomenon does not arise however much the stimulation has been prolonged. For instance, in the experiments certain recordings of which are presented in Fig. 28 it was established that during several-minute stimulation at a rhythm of 1 per second the effect of its character did not change (osc. A). (Legend to Fig. 28, textpage 67: The arising of positive potentials in connection with increase of frequency of stimulation of the surface of the cortex. Cat No. 56, July 16, 1953. Nembutal. The stimulating electrodes and discharge electrode are established on the surface of the gyrus suprasylvius;

distance between discharge electrode and stimulating electrodes 5 mm.
Intensity of stimulation 30 v. A - frequency of stimulation about 1
per second, B - about 5 per second, C - about 10 per second. D - about
25 per second; 0.5 second after the beginning of stimulation. E - 10
seconds after beginning of stimulation. Recording by Schleiff oscillo-
graph.) At a frequency of stimulation of 5-10 per second, in spite
of the fact that complex double negative potentials arose, i.e in
spite of the excitation of certain complexes of intermediate cortical
neurons, no change occurred of the sign of the biopotential; at
prolonged stimulations progressive attenuation occurred of a supple-
mentary fluctuation (osc. B and C). At a frequency of stimulation
of 25 per second the negative potentials began quickly and progressively
to attenuate (osc. D), and after several seconds of stimulation each
shock provoked a quite positive potential (osc. E).

In certain preparations a powerful single stimulation can
immediately provoke a positive potential of greater amplitude, arising
after the artefact of stimulation. It is interesting that in these
cases at a frequency of stimulation of 10 per second only the first
shock of stimulation provokes a positive potential; the second and
all subsequent shocks of stimulation provoke negative potentials
(Fig. 22, C).

Positive potentials, considerable in amplitude, can arise

after a supplementary negative potential (Fig. 28, A and B).

According to Chang, preceding a supplementary negative potential is a positive one, i.e. in response to stimulation a negative potential arises that passes over into a positive one, after which a second negative fluctuation follows. This actually can occur (see Fig. 15), but this is not the rule: supplementary negative fluctuations can arise without the preliminary first negative potential having changed to positive (see Fig. 22).

Finally, a positive fluctuation (Fig. 23, C) can precede the initial negative potential, which Chang (1951) also points out.

In the majority of cases after the negative potential a low positive deflection is observed of considerable length. It is difficult to say whether it always expresses activation of the elements of the deep layers, since it is observed also during deep narcosis. Perhaps this is the analogue of the subsequent positive potential after regional excitation. Such resultant potentials arise, as already said, in the motoneurons.

The following data regard the positive potentials that arise at stimulation of the surface of the cortex.

1. In experiments on isolated strip of cortex the following facts were established. When the cortex was not narcotized, then in response to a shock of stimulation applied to the surface of the cortex a brief negative potential arises (30 milliseconds), after

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which a positive potential follows that lasts 2-4 seconds; on its

background fluctuations arise at a frequency of 60-75 per second.

At light narcosis these fluctuations drop out and a long positive

potential arises in pure form (Burns, 1951). During deep narcosis

the length of the positive potential is shortened to 0.1 second,

i.e. in response to a single stimulation the two-phase effect already

described arises: after the negative potential a positive one arises

of less amplitude and greater length (see, for instance, Fig. 8).

At sinking the discharge electrode deeper into the cortex the

positive potential discharged from the surface of the cortex changes

its sign at a depth of 0.4 mm. Thus, that the positive potential is

connected with the active state of the elements found below layer II

of the cortex is directly demonstrated (Burns, 1951). At the micro-

electrode being sunk in deeper, the positive potential attenuates,

then changes its sign, i.e. a negative potential is registered. The

latter reaches its greatest amplitude at a depth of about 1.3 mm.

It is characteristic that the amplitude of the negative potential

at this depth exceeds the amplitude of the positive potential

discharged from the surface of the cortex (compare recordings a and

d, Fig. 29, textpage 69, the legend to which reads: Bioelectrical

potentials discharged by the microelectrode from different layers

of the cortex at stimulation of its surface. Isolated strip of

cortex of cat with circulation preserved. The glass microelectrode

was 4 mm. distant from the stimulating electrodes, and later on it
was sunk more deeply into the cortex. The indifferent electrode
was placed on a thermocoagulated part of the cortex. The surface
of the cortex was stimulated with a single electrical stimulus.
Booster with a more fixed time. a - Microelectrode on the surface
of the cortex; b - microelectrode is sunk to a depth of 0.59 mm.;
c - at 0.72 mm.; d - at 1.5 mm.; e - at 2.03 mm. Deflection upward
signifies the negative character (Burns and Grafstein, 1952).).
Thus, the main source of a positive potential discharged from the
surface of the cortex at stimulation of the surface of the cortex
consists of neuronic elements located at a depth of 1.3-1.5 mm.,
i.e. neuronic elements of cortical layers V and VI. The fact that
the threshold of provocation of the positive potential is least
at the position of the tip of the stimulating microelectrode at this
depth, on the other hand, testifies to this. It is greatest when
the stimulating electrode is found on the surface of the cortex
(Burns and Grafstein, 1952).

2. While stimulation of the motor region of the cortex causes
negative biopotentials, contraction of the corresponding muscles does
not set in. Movements arise only when stimulation of the cortex
begins to cause positive potentials, contraction of the muscles
setting in when the positive potentials discharged from the surface
of the motor region of the cortex reach a certain definite magnitude.
Thereby, the group of impulses of excitation in the muscle (Adrian,

1936) corresponds to each positive fluctuation in the cortex. At
the time the positive potential is discharged from the surface of
the cortex discharges of very quick impulses of larger amplitude
are discharged from the depths of the cortex by a microelectrode.
These discharges are registered only when the tip of the electrode
is at a depth of 1.3-1.5 mm., i.e. in the region of the main source
of the positive potential, in the region where the bodies of the
larger pyramids of layer V are found (Burns and Grafstein, 1952).
These discharges, as already said, never arose at the time of a
negative surface potential. Thus, the positive potential registered
from the surface of the cortex is connected with excitation of the
pyramidal neurons of the deep layers of the cortex.

3. Positive response spreads without decrement at a rate of
0.6-0.15 m. per second (Adrian, 1936; Burns, 1951). During narcosis
it spreads for a distance of up to 7.5 mm. On the basis of the
spread without decrement, the conclusion is made that the spread
of positive potentials is linked with transmission of excitation
into the deep layers of the cortex from neuron to neuron through
the synapses (Adrian, 1936). The spread of the positive potential
is avoided by incision of the cortex to a depth of 1.25 mm. (Burns
and Grafstein, 1952). According to Burns, neurons of the deep
layers form a network consisting of "self-excitation" nerve orbits.

The minimal length of these nerve orbits, in his opinion, equals 2 mm. (Footnote: In a 1951 paper Burns gives a graphic depiction of this network. In the illustration of the Burns and Grafstein article (1952) the elements of the network are presented in the form of cells with offshoots; However, the depiction given there of the connections of the neurons is senseless from the point of view of generally known neurological data: the offshoots of the neurons establish synaptic connections between one another, i.e. the axon of one neuron with the axons of other neurons, the dendrites of one neuron with the dendrites of other neurons. Finally, in the 1955 work Burns supports some of his theoretical ideas on the activity of the network of nerve elements of the cortex by model experiments with "titration neurons" (!).)

Origin of positive potentials arising during stimulation of the surface of the cortex. On the basis of a number of facts cited above it is possible to consider demonstrated that the positive fluctuations discharged from the surface of the cortex during stimulation of its surface are connected with excitation of the neuronic elements of the deep layers of the cortex. On the basis of electrophysiological and histological facts known at the present time it is possible to explain first the arising of positive potentials during excitation of the elements of the deep layers and, secondly, it is possible to explain in what way during stimulation

of the surface of the cortex excitation of the elements of the deep
layers arises.

During clarification of the arising of the positive potential
discharged from the surface of the cortex it is possible to use
the same reasoning as for clarification of the arising of a positive
potential discharged from the depths of the cortex at activation of
the surface layers of the cortex, i.e. it is necessary to think
that when regional excitation, a negative potential, arises in the
bodies of the pyramidal neurons, then the corresponding top dendrites
of these pyramids must be polarized in a positive fashion. This is
virtually observed too, and this is directly shown by Burns's
experiments. As already said, at sinking the discharge electrode
into the cortex to a depth of 0.4 mm. and lower, a negative potential
is registered (the same electrode discharges a positive potential
when on the surface). Finally, it is known that at direct stimulation
of the neuronic elements of the deep layers (by means of a needle
electrode inserted deep into the cortex) from the surface of the
cortex positive fluctuations of potential are discharged (Adrian,
1936; Burns and Grafstein, 1952).

Thus, the positive potential discharged from the surface of
the cortex expresses a positive polarization of the top dendrites
of the pyramidal neurons, stipulated by regional excitation of the
deeply situated parts of these neurons, probably of their cellular
bodies for the most part.

During very intense stimulations of the cortical surface the
loops of electrical current can show a direct stimulating action on
the neuronic elements of the deep layers and cause their excitation.
However, during the conditions of stimulation which were used in
my experiments, as already said repeatedly, primarily the fibers of layer
I were excited. Excitation of layer I fibers can stipulate excitation
of deep layers by neurons of layer I, on which collaterals of layer I
fibers end. Since the descending axons of the (modified) star pyramids
of layer II discharge collaterals into layers III, V, and VI and certain
neurons of layer II have an axon terminating around the bodies of the
pyramidal neurons of layer III, then consequently at excitation of
these neurons activation of the deep layers should occur. If the
neurons of layer II with horizontal axon (Fig. 26 - 2) are excited,
the collaterals of which terminate in many star pyramids of layer II,
then this can evidently lead to excitation of a whole complex of the
latter and to most intense "impulsation" into the deep layers of the
cortex.

The following facts obtained by Burns and Grafstein (1952)
speak in favor of this, that excitation of the neurons of the deep
layers and the arising of the positive potential can proceed and
occur as a result of the excitation of the fibers of layer I. When
the intensity of stimulation of the cortical surface was threshold

in the sense of the provocation of a positive potential at a certain

distance from the region of stimulation, then surface-most section

of the cortex around the stimulating electrodes, which stopped the

spread of the negative potential, inhibited the arising of the

positive potential too. During intense stimulations of the surface

of the cortex the positive potential spread even after intersection

(again the depth of the section, as already stated, did not reach

1.25 mm.), but the latent period of its arising in a remote part

after surface section of the cortex was lengthened several times.

Thus, excitation of the fibers of layer I can stipulate the arising

of a positive potential. By means of the fibers of layer I

excitation of the deep elements of the cortex in a part remote

from the region of stimulation can proceed more quickly than when

the elements of this part are activated as a result of the successive

spread of the excitation through the neuronic elements of the deep

layers of the cortex.

Thus, with stimulations of the cortical surface moderate in

intensity excitation of the elements of the deep layers occurs through

the fibers of layer I that are first to be excited.

According to Burns (1951), the negative potential arising

immediately in response to stimulation is the reason for the arising

of a positive potential. When the negative potential reaches a certain

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critical magnitude, namely 30-35% of its maximal amplitude, then it

becomes the source of a discharge of impulses which spread downward

through a top dendrite to the body of the pyramidal neuron. Spreading

through the axon of the pyramidal neuron and its collaterals, the

excitation embraces other neurons of the deep layers. Eccles (1951)

thinks this the most likely explanation of the mechanism of the arising

of positive potentials. Chang (1951) also thinks that the positive

potential arises when the impulses of the top dendrites spread to

other parts of the pyramidal neurons.

Thus, with such integration the local potential of dendrites of

the pyramids is considered as a source of the excitation of the pyramidal

neuron. We, with this point of view, run into the diametrically opposite

one which is developed in the present work and according to which

regional excitation of the dendrites is not the source of the excitation

of the corresponding neuron.

Adrian came to the conclusion that the arising of the positive

potentials being considered is the result of the fact that the bodies

of the pyramidal cells are excited and that their dendrites are at this

time in an inactive state (Adrian, 1938). However, it is impossible

to allege that at electrical stimulation of the surface of the cortex

elements of the deep layers, particularly of the body of the pyramids,

were selectively excited. It must be thought that in this case too,

when the positive potential is registered from the surface, elements

of both deep and surface layers are found in an active state, however

the activity of the deep layers is dominant, and it must be thought

that the potential being registered from the surface of the cortex is

the result of a negative potential and a positive potential. At

stimulation of the cortical surface, owing to the spread of excitation

through the system of fibers of layer I, the top dendrites of a certain

number of pyramidal neurons of the point of the cortex being discharged

come into the regional excitation. However, at excitation of the

fibers of layer I excitation can also occur of the deep layers through

the star and other neurons of layer II. As a result of this, the deep-

lying parts of the pyramidal neurons, of their bodies, can enter into

a state of regional excitation. If, for simplicity, one pyramid neuron

is taken, then a regional excitation in the summit dendrite arises

in it under the influence of impulses from the first source under a

certain number of synaptic endings of the fibers of layer I. If at

this time excitation from a second source of a greater number of

synapses occurs for the body of the neuron being considered, then a

more intensive regional excitation arises there and the negative

dendritic potential will be masked by a positive polarization of the

dendrite, stipulated by the negative potential of the body of the

pyramidal neuron. On the other hand, the negative potential too,

discharged from the surface of the cortex, should not always testify

to the isolated activation of the elements of the surface layers:

regional excitation of the dendrites can mask regional excitation of

the bodies of the corresponding neurons. Hence, logically it follows

that, with more or less identical excitation of the surface and deep

layers, from the surface of the cortex as a result of an algebraic

summation of potentials identical in intensity and opposite in sign

there can generally be registered a certain electrical potential.

During deep narcosis the sign of the potential discharged from the

surface of the cortex can actually testify to the activation of the

surface layers (during stimulation of the surface of the cortex) or

of the deep layers (at stimulation of the afferent fibers - see

Chapter IV). This is connected with the fact that during deep

narcosis activity is limited by those elements on which impulses

from the fibers of layer I that are stimulated or the afferent

impulses act directly. In these elements regional excitation arises,

and the reaction terminates in this. At light narcosis when the

excitability of the neurons is relatively high subsequent excitation

of the neurons occurs, as well as transmission of the excitation by

numerous elements in the various layers of the cortex and, as we

see further on, in different parts of the cortex. Certainly an

analysis of the bioelectrical potentials being registered from the

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surface of the cortex becomes in connection with this much more
complex.

In connection with what has been said above, certain recordings
of bioelectrical potentials will be considered below.

We have seen that at deep placement of the discharge electrode
below layer II the negative potential arising immediately in response
to stimulation changes its sign: the negative potential is recorded
from the surface, the positive potential from the depths. As for
the supplementary negative potential, as Chang (1951) ascertained,
its sign does not change at deeper placement of the electrode. This
is seen in recordings C and D of Fig. 15. A supplementary negative
potential expresses regional excitation of the top dendrites arising
under the effect of impulses from the neurons of layer II that are
excited. However, as we have seen, at excitation of the neurons of
layer II a transfer of impulses occurs to elements not only of the surface
but also of the deep layers of the cortex, as a result of which the
discharge electrode found in the deep layers discharges a negative
potential of the excited neuronic elements with which it comes in contact.
Thus, under the influence of impulses from the fibers of layer I an
isolated activation arises of the elements of the surface layers. At
excitation of the intermediate neurons of layer II activation occurs
of the elements both of the surface and of the deep layers of the cortex.

In Fig. 30 are presented recordings interesting to analyze.
With a stimulation intensity of 16 v. (threshold 3 v.) shocks of
stimulation at a rhythm of 8 per second caused double negative
potentials that set in without an appreciable latent period (osc.
A). At switching to a stimulation frequency of 40 per second each
shock of stimulation began to provoke a negative potential of
considerably lesser amplitude than at infrequent stimulations. The
second negative fluctuation was especially reduced. After several
seconds of stimulation the effects attenuated still more. At
switching to a frequency of stimulation of 8 per second the stimulation
shocks began to provoke the same effects as prior to tetanizatinn
(osc. B). With the intensity of the stimulation at 30 v. the stimulation
shocks at a rhythm of 8 per second caused effects considerably different
from the effects of stimulation at 16 v. (osc. C): insignificant
negative deflection arose even prior to its completion 6 milliseconds
after the moment of stimulation, and a powerful negative potential
set in with additional fluctuations in its descending part. Thus,
the impression is created (if the initial first insignificant fluctua-
tion is mistaken for an expression of polarization from the intense
electrical stimulation) that at energetic stimulation an effect arises
with a greater latent period. However, there are no bases to consider
the initial deflection as a whole an artefact. It is necessary to

consider it a highly attenuated negative potential, which at 16 v.

reached a considerably greater amplitude. It is possible to explain

this phenomenon by the admission that energetic stimulation causes

activation of the elements of the deep layers (perhaps because of

the fact that the current directly excites not only the fibers of

layer I but also the cells of layer II). The positive potential

arising at activation of the deep elements almost removes the negative

potential which the elements of the surface layers develop. The

results of experimentation with a change-over of the frequency of

stimulation to 40 per second speak in favor of the accuracy of such

a hypothesis. After 13 shocks of stimulation purely positive potentials

of low amplitude and length began to arise. After switching again

to infrequent stimulations the effects gradually became such as they

were prior to tetanization. It is possible to observe the change-over

from purely positive initial fluctuations to insignificant negative

initial fluctuations (osc. D, E).

(Legend to Fig. 30, textpage 74: Arising of positive potentials

in connection with intensification and increase of frequency of stimula-

tion of the cortex. Cat No. 27, June 7, 1950. Nembutal. Stimulating

electrodes and the discharge electrode are placed on the gyrus

suprasylvius. Distance between discharge electrode and stimulating

electrodes = 3 mm. A - intensity of stimulation 16 v., frequency in

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the beginning at 8 per second, then 40 per second. B - end of one-second stimulation with frequency of 40 per second and shift to a frequency of 8 per second. C - intensity of stimulation 30 v.; frequency in the beginning at 8 per second, then 40 per second. D - end of one-second stimulation with a frequency of 40 per second and a shift to a frequency of 8 per second. E - continuation of recording D.)

(Legend to Fig. 31, textpage 75: "Conversion" of positive potentials into negative in connection with prolonged stimulation of the cortex. Cat No. 58, Nov. 21, 1953. Booster with more constant time. Stimulating and discharge electrodes are placed on the gyrus suprasylvius. Bipolar discharge: one discharging electrode is found at a distance of 5 mm, the second at a distance of 15 mm. from the stimulating electrodes. The intensity of stimulation 30 v. Frequency of stimulation about 5 per second. A - beginning of stimulation. B - 0.7 second after A. C - effect of stimulation after prolonged stimulation of the brain.)

In oscillogram A of Fig. 31 is presented the effect of the beginning of stimulation of the surface of the cortex at a rhythm of 5 per second. After the artefact of stimulation a negative potential arose which was quickly broken and a positive deflection of greater amplitude developed. As a result of prolonged stimulation the

positive potentials ceased arising, and the stimulation began to

cause only negative potentials (osc. C). It is characteristic

that the latter now had greater amplitude than when the complex

positive potentials arose after them. It is evident that activation

of the deep layers of the cortex masked the activation of the elements

of the surface layers.

Thus, we have encountered complex bioelectrical phenomena.

Each recording requires special analysis, and in the conclusions

to which the analysis leads there sometimes remains much of the

hypothetical.

4. Contribution to the Question of the Spread of

Activity Through the Cerebral Cortex

Below a number of recordings will be considered that were

obtained in experiments with stimulation of the cortex and registration

of the biopotentials, which were made on animals under very light

narcosis. These data compel one to recognize that the position,

thanks to which the negative potentials that arise at direct stimulation

of the cortex spread with decrement for moderate distances, is in need

of major correctives.

(Legend to Fig. 32, textpage 76: Distribution of activity

through the cerebral cortex from the point stimulated. Cat No. 27,

June 7, 1950. Relatively shallow nembutal narcosis. Stimulating

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electrodes (P) are placed on the anterior pole of the gyrus
suprasylvius. In experiments A-D the potentials are discharged
simultaneously from point E_1 (upper curves) at a distance of 3 mm.
from P and from point E_2 (lower curves) at a distance of 13 mm.
from P. A - effect of stimulation during an intensity of stimuli
of 16 v. and a frequency of 10 per second. B - aftereffect. C -
beginning frequency of stimulation 10 per second, then the frequency
changes to 50 per second. D - intensity of stimulation 30 v.,
frequency 10 per second. In experiment E the second discharge
electrode E_2 is placed on the posterior pole of the same gyrus.
Distance of P - E_2 = 21 mm.; frequency of stimulation 10 per
second, intensity - 16 v.)

The recordings of Fig. 32, A - D, were obtained in the following
way. On the gyrus suprasylvius were placed: the stimulating pair
of electrodes (P) and, at a distance of 3 and 13 mm. from them,
the discharge electrodes (E_1 and E_2). The threshold of provocation
of the negative potential at point E_1 equaled 3 v. In order for
the negative potential to arise at E_2 the intensity of stimulation
necessary was 10 v. With 16 v. at point E_2 considerable potentials
arose but of 4 times lesser amplitude than at point E_1 (osc. A). At
point E_1 the negative potentials arose with an insignificant period
of latency. At point E_2 it was with a latent period of 10-11 milliseconds,

which assumes a rate of spread of about 1.5 m. per second.

"Spontaneous" negative potentials arose at both points
simultaneously (osc. B).

Thus, as a result of the stimulation of one point of the
gyrus suprasylvius activation occurs of the top dendrites of the
pyramid neurons over a wide territory of this convolution.

(Legend to Fig. 33, textpage 77: Spread of activity
through the cerebral cortex from the point being stimulated.
Cat No. 36, Jan. 2, 1951. The potentials are discharged simultaneously
from the middle part of the gyr. suprasylvius (E_1, upper curves) and
from the gyr. sigmoideus post. (E_2, lower curves). The first pair
of stimulating electrodes (P_1) is placed on the anterior pole of gyr.
suprasylvius, the second pair (P_2) on the posterior pole of this
convolution (see scheme). The distance P_2-E_2= 30 mm. A - "spontaneous"
activity. B and C - stimulation through P_1. B - effect of the 20th
shock at a frequency of 2 per second and an intensity of 12 v. C -
intensity of stimulation 25 v.; at first the effect of one shock, then
a stimulation of a frequency of 10 per second is applied (attention
is drawn to the fact that a single stimulation was applied at the time
of the "spontaneous" slow fluctuation in gyr. suprasylvius). D - effect
of stimulation through P_2.)

Attention is called to the fact that with a frequency of
stimulation of 50 per second at a remote point potentials ceased to

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arise in response to shocks of stimulation and at the most proximal

point for each shock of stimulation a negative potential arose

(osc. C), i.e. the impression was created that at a stimulation

frequency of 50 per second transmission of excitation at point E_2

ceased.

At stimulation of the anterior and middle parts of the gyrus

suprasylvius negative potentials arise in the posterior gyrus

sigmoideus. In the same preparation stimulation of the posterior

pole of the gyrus suprasylvius cannot lead to the arising of bio-

potentials in the gyrus sigmoideus (Fig. 33).

(Legend to Fig. 34, textpage 78: Spread of activity through

the cerebral cortex from the point stimulated. Cat No. 29, June

28, 1950. The stimulating and 2 discharging electrodes are placed

on the surface of the gyrus suprasylvius. The first discharge

electrode (E_1) is at a distance of 3 mm., the second (E_2) at a distance

of 14 mm. from the stimulating electrodes. The biopotentials discharge

simultaneously from points E_1 (upper curves) and E_2 (lower curves).

A - intensity of stimulation 30 v., frequency 10 per second, beginning

of stimulation. B - intensity of stimulation 20 v., frequency 10

per second. C - frequency 20 per second. D - 100 per second.

Experiment E was carried out prior to experiments A - D; intensity

of stimulation 30 v., frequency 10 per second; at time of exposure

the direction of the stimulating current was changed (attention is

called to the direction of the artefact of stimulation).)

With very light narcosis (Fig. 34) the activity begins to
spread from the point being stimulated not only to greater distances
but also without appreciable decrement: the amplitude of the potentials
at a remote point can be even greater than nearby (osc. A and B).
Furthermore, complex effects arise, the character of which shows that,
together with impulses from fibers of layer I by direct activation of
the top dendrites, additional activation of them occurs from the
intermediate cortical neurons, the various complexes of which are
included in the activity at a different time. A small positive
fluctuation precedes the negative potential at point E_2. However,
this is evidently an expression of polarization, since this fluctuation
disappears at change of direction of the stimulating current, whereas
the character of the negative potential is not changed (osc. E).

Sometimes just one shock of stimulation causes prolonged
rhythmical aftereffect. In the aftereffect the activity spreads
through the cortex for a greater distance. In the experiment, the
recordings of which are presented in Fig. 35, the shock of stimulation
applied to the surface of the anterior pole of the gyrus suprasylvius
caused no bioelectrical reaction at point E_2 (19 mm. from the place
of stimulation), an energetic two-phase potential (osc. B) having
been provoked at point E_1 (4 mm. from the place of stimulation). In

the aftereffect the activity gradually took hold of this remote
point (osc. B and C).

(Legend to Fig. 35, textpage 79: Spread of activity through
the cerebral cortex from the point stimulated. Cat No. 21, May 10,
1950. The stimulating pair of electrodes and 2 discharge electrodes
were placed on the surface of the gyrus suprasylvius. The first
discharge electrode (E_1) was at a distance of 4 mm. and the second
(E_2) was at a distance of 10 mm. from the stimulating electrodes.
The biopotentials discharged simultaneously from point E_1 (upper
curves) and point E_2 (lower curves). A - "spontaneous activity".
B - effect of one shock of stimulation; intensity of it 30 v.;
length of stimulating stimulus 0.5 millisecond. C - continuation of
B recording.)

It is possible to think that at the point of the cortex
stimulated the cortical neurons excited by the stimulation rhythmically
continued to be excited and evidently the number of neurons excited
gradually increased on the strength of summation.

At stimulation of the cortical surface at a rhythm of 10 per
second a bioelectrical reaction at a remote point can set in after
prolonged stimulation and then when the stimulation is not stopped it
can continue to be intensified (Fig. 36, A and B). Certainly the
spreading of the activity occurs in these conditions because of the

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excitation of an entirely greater number of cortical association neurons. It is characteristic that when in the experiment being considered powerful negative potentials began to be registered from a remote point, then the effect at the nearest point became more complex too: additional negative fluctuations appeared (osc. B).

(Legend to Fig. 36, textpage 80: Spread of activity through the cerebral cortex from the point stimulated. Cat No. 29 (i.e. the same preparation as for Fig. 34). The electrodes were placed on the gyrus suprasylvius, but they were all shifted frontward. $P-E_1 = 4$ mm.; $P-E_2 = 14$ mm. The potentials were discharged from point E_1 (upper curves) and point E_2 (lower curves). A - intensity of stimulation 30 v., frequency 10 per second. B - after 10 seconds of stimulation. C - frequency of stimulation was changed from 10 to 50 per second.)

At increase of frequency of stimulation up to 50 per second (osc. C) the transmission of excitation shifted to remote point E_2, and at the proximal point E_1 the potentials right after the first shocks of frequent stimulation changed their sign, which assumes intense excitation of the elements of the deep layers of the cortex.

Attention is attracted to the fact that when, as a result of prolonged stimulation, at a remote point of the cortex slow negative biopotentials arise, then there is no appreciable difference in the latent periods of the arising of slow potentials at the proximal

(4 mm.) and remote (14 mm.) points. Thus, the spread of excitation
can proceed at a very great rate.

Evidently at prolonged stimulation certain cortical neurons are
excited, the axons of which proceed for greater distances and end
mainly in the dendritic surface layers of the cortex. The excitability
of these neurons rises in such measure that, in response to stimulation,
they are immediately excited and, if it is assumed the axons of these
neurons conduct the excitation at a higher rate, then it is possible
to explain the absence of appreciable differences in the latent periods
of the arising of bioelectrical effects at points E_1 and E_2.

The bioelectrical potentials at stimulation of the gyrus
suprasylvius are registered, as already said, in the gyrus sigmoideus.
Figs. 33 and 37, A (upper curves) illustrate this. However, at
stimulation of the gyrus suprasylvius considerable biopotentials can
be registered also from the gyrus ectosylvius (Fig. 37, A, lower
curves). According to the morphological data of Bekhterev, this
spread of excitation from the gyrus suprasylvius to the gyrus
ectosylvius must proceed through the fibers in the upper part of
layer I, which serially connect the gyri lying parallel in a line.

A peculiar reaction is observed at the posterior pole of the
gyrus suprasylvius at stimulation of the middle or anterior parts
of this gyrus: from the surface of the posterior part of this gyrus

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positive fluctuations of a bioelectrical potential are discharged

(Fig. 32, E; see also Fig. 38, C). Evidently, as a result of the

stimulation, excitation occurs of the fibers that terminate in the

posterior part of the gyrus mainly in the deep layers of the cortex.

(Legend to Fig. 37, textpage 81: Spread of activity through

the cerebral cortex from the point stimulated. Cat No. 27, June

7, 1950. A - Stimulating electrodes are placed in the anterior part

of the gyr. suprasylvius; the potentials are discharged from the

gyr. sigmoideus post. (upper curve) and from the gyr. ectosylvius

(lower curve). The intensity of the stimulation was 30 v., the

frequency 10 per second. A_1 - course 1 second after A. B - stimulating

electrodes on the gyrus suprasylvius; potentials are discharged from

the same gyrus at a distance of 4 mm. from the point stimulated (upper

curve) and from the point of the gyrus suprasylvius of the opposite

hemisphere symmetrical to that which was stimulated (lower curve).

Intensity of stimulation 30 v., frequency 10 per second.)

As Danilevskii ascertained, stimulation of any point of one

hemisphere leads to the arising of a biopotential at a symmetrical

point of the opposite hemisphere. According to Chang's data (1953),

the part of the cortex activated in the opposite hemisphere is no

more than 4 mm^2. in area. In Fig. 37, B (lower curves) are shown

the effects in the opposite hemisphere at a point symmetrical to

that stimulated. These characteristic "callosal effects" (Curtis,

1940) are two-phase potentials similar to those that arise at peripheral stimulations, i.e. after the positive phase a negative follows. Fig. 38 shows that the negative phase cannot arise, and in such cases the "collosal effect" is expressed only by a positive fluctuation.

(Legend to Fig. 38, textpage 82: Activity spread, from point stimulated, through the cerebral cortex. Cat No. 20, April 28,1950. In all the experiments one and the same point of the anterior pole of the gyr. suprasylvius is stimulated; the intensity of the stimulation is 25 v.; the duration of the irritating stimulus is 0.5 millisecond. The first discharge electrode (E_1) is placed on the gyr. suprasylvius at a distance of 7 mm. from the stimulating electrodes (P); in all oscillograms the lower curve consists of potentials from E_1. The second discharge electrode is placed on different parts of the cortex: A - on the gyr. ectosylvius, its anterior pole; B - on the gyr. ectosylvius, its posterior pole; C - on the posterior pole of the gyr. suprasylvius; D - on the gyr. suprasylvius of the opposite hemisphere at a place symmetrical to the place of stimulation; E - on the posterior pole of the gyr. suprasylvius of the opposite side; F - on the posterior pole of the gyr. ectosylvius of the opposite side.)

At relatively light narcosis considerable positive potentials arise in response to stimulation of the cortex and in asymmetrical parts of the corresponding gyrus of the opposite hemisphere and even

in noncorresponding gyri. For instance, in experiments the recordings
of which are presented in Fig. 38, E and F, at stimulation of the
anterior pole of the gyrus suprasylvius the bioelectrical potential
was registered from the posterior pole of the opposite gyrus suprasylvius
and from the posterior pole of the opposite gyrus ectosylvius. Finally,
these effects arose also through the callosal fibers that guarantee
the connection of the two hemispheres, but the manner of this connection
was not so simple as was depicted by Chang: this is not a connection
of one with the other of only precisely symmetrical parts. Evidently
Cajal's idea was correct (cited by Bekhterev, 1898), that the fibers
of the corpus callosum through the collaterals going out from them
link together, besides the symmetrical parts of the hemispheres, the
many other cellular elements also that are situated in the most diverse
cortical layers and areas.

(Legend to Fig. 39, textpage 83: Activity spread through the
cerebral cortex from the point stimulated. A and A_1 - cat No. 21,
May 10, 1950. Stimulating electrodes and two discharge electrodes are
placed on the surface of the gyrus suprasylvius; the first discharge
electrode (E_1) is 4 mm. distant, and the second (E_2) is 20 mm. distant
from the stimulating electrodes. Biopotentials are discharged
simultaneously from point E_1 (upper curves) and from point E_2 (lower
curves). The intensity of stimulation is 30 v., the frequency 10 per

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second. A - beginning of stimulation; A_1 - several seconds after stimulation. B and B_1 - cat No. 22, May 12, 1950. Location of electrodes is shown in the scheme. B - beginning of stimulation (25 v, 10 per second); B_1 - several seconds after stimulation.)

Recordings A and A_1 in Fig. 39 show constant involvement in the reaction of a point of the cortex located 20 mm. distant from the point irritated. The irritating electrodes were set up on the anterior pole of the gyrus suprasylvius and the discharge electrodes at a distance of 4 and 20 mm. from them. The first 3 shocks of stimulation caused bioelectrical reaction only at the nearest point of the cortex (osc. A); then each shock of stimulation began to provoke slow negative potentials both at the nearest point and at remote points (osc. A_1). Attention is attracted to the fact that potentials at a remote point have been altered in regard to amplitude, form, and the latent period of their setting in. Undoubtedly in this case the activity spread occurred because of excitation of the association neurons.

Recordings B and B_1 of Fig. 39 are complex for analysis. Stimulation of the gyrus suprasylvius at a rhythm of 10 per second did not provoke for a long time bioelectrical reactions in the analogous convolution of the opposite hemisphere (osc. B, lower curve), but then ever-intensifying negative potentials arose there,

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the amplitude and duration of which even exceeded the amplitude and

length of potentials discharged from the cortex in the sphere of

stimulation (osc. B_1); the rhythm of the potentials in the opposite

hemisphere corresponded to the rhythm of stimulation, but they arose

with a very different latent period and were very altered in form,

amplitude, and length. It is possible to think that the bioelectrical

reaction described was governed by the activity of the intermediate

cortical neurons of the opposite hemisphere that were gradually

implicated in the reaction under the effect of impulses from the

callosal fibers. (Footnote: It has been ascertained that strychnine

has almost no influence on the characteristic "callosal effect", i.e.

on the complex of initial positive and, following after it, negative

potential. Under the action of strychnine an energetic negative

potential arises after the effect described (Chang, 1953), i.e.

under the action of strychnine the supplementary, often invisible,

fluctuations that set in after the characteristic callosal effect

are extremely intensified. Evidently at prolonged stimulation of

the opposite hemisphere elevation occurs of the excitability in these

neurons (which are sensitive to strychnine), and they are excited,

stipulating the arising of powerful negative slow potentials. Evidently

from moment to moment their degree of excitability is changed, which

stipulates the alterability of the bioelectrical reactions.)

Thus, just a single electrical stimulation of any point of the

surface of the cortex can cause an answering bioelectrical reaction at

actually any other point of the cortex. Moreover, in the corresponding

hemisphere there arise mainly negative biopotentials, which indicates

the activation or chief activation of the neuronic elements in layer I

and II.

In the opposite hemisphere by means of the callosal fibers

there occurs, first of all, activation of the elements of layer III

(chief place of the endings of the callosal fibers), which is

expressed in the arising of a short-term positive potential. However,

then, when the functional state of the cortex is good a negative po-

tential arises, i.e. activation occurs of the elements of the surface

layers.

Thus, just one shock of stimulation applied to the surface of

the cortex provokes activation of a great number of neuronic elements

of the cortex.

Excitation spread from the point stimulated occurs first by

a system of directly stimulated fibers of layer I, secondly by a

system of callosal fibers excited by the stimulation, and thirdly by

means of the intermediate and association neurons of the cortex. In

the last case the reaction in the remote parts sets in often only

after prolonged stimulation by a constant involvement in the reaction

(on the basis of constant elevation of the excitability) of the

intermediate neurons under the action of impulses from the fibers

stimulated in layer I (or under the effect of the stimulating

current at the place of stimulation). When the bioelectrical

reaction at a remote point is stipulated by the activity of the

intermediate neurons, then the amplitude of the biopotentials

arising there may be greater than the amplitude of the potentials

arising in immediate proximity to the part stimulated.

The electrographic data presented illustrate the known

position of irradiation of excitation through the cerebral cortex.

In the case in question a particular case of the irradiation

of the excitation provoked by direct stimulation of the surface of

the cortex was analyzed. However, certainly the data obtained

offer interest in general for understanding the phenomenon of

irradiation that lies at the base of the most important mechanisms

of the activity of the cortex.

On the other hand, it has been shown that the irradiation of

the excitation provoked by stimulation of layer I of the cortex leads

to the arising of chiefly negative potentials in the vast cortical

territories, i.e. to activation of the surface layers of the cortex;

in other words, at excitation of a certain number of fibers in layer

I the top dendrites of the pyramidal neurons in the vast territory of

the cerebral cortex can enter into a state of regional excitation.

Since the fibers of layer I are mainly axons of the neurons with
axon rising from all layers of the cortex and recurrent collaterals
of the pyramidal neurons from all layers of the cortex, then a
priori it is possible to conclude that at excitation of a certain
number of these neurons an effect can set in similar to that which
is obtained at direct electrical stimulation of their axons: the
arising over the range of almost the whole cortex of a regional
excitation of the top dendrites of the pyramidal neurons of the
cortex. What is the physiological meaning of this phenomenon?
If it is assumed that the regional excitation of the dendrites
stipulates the inhibition of the corresponding neurons, then the
phenomenon considered acquires special interest.

In Beritov's opinion the main function of layer I consists
of the making of a general inhibition of the entire cortex. Layer
I unites the whole cortex in regard to inhibition (Beritov, 1953).
On the other hand, as we saw, excitation of the fibers of layer I
can provoke activation and excitation of the elements of the deep
layers and stipulate motor reaction. Evidently the excitation of
the system of fibers of layer I, provoking the inundation oppression
of the excitability of the cortical elements, can adapt to the
excitation and fortify it in certain complexes of the cortex found

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in a state of elevated excitability. In Chapter III we encounter

facts that speak in favor of this last hypothesis.

5. Supplement: Concerning the Phenomenon of the Slowly

Spreading Depression of Cortical Electrical Activity

Since during experiments with direct stimulation of the cortical

surface it is impossible not to pay attention to the phenomenon of

the slow-spreading depression of electrical activity discovered by

Leao (1944), it will be of value to give in the form of a supplement

to this chapter a survey of the facts published and of the hypotheses

expressed regarding this phenomenon.

The phenomenon of the slow-spreading depression. If stimulating

electrodes are placed on the surface of the cerebral cortex of rabbit

and the discharge electrodes at a distance of 1-2 mm. from them, then

after several seconds of tetanization by a relatively weak induction

current at once, or after 10-20 seconds, a progressive attentuation

of the "spontaneous" electrical activity sets in. In the last case

from the moment of cessation of stimulation to the beginning of the

development of depression the "spontaneous" electrical activity has

a normal character. At the time of a state of depression the electrical

activity in some cases simply attenuates sharply; in other cases it

not only attenuates but also changes its character in the sense of

reduction of the frequency of the bioelectrical fluctuations. Finally,

on the background of the depression of the electrical activity

fluctuations may arise of a spasmodic character. Restoration of the

normal picture of electrical activity occurs after approximately 10

minutes (Leao, 1944).

Depression of the bioelectrical activity gradually spreads in

all directions from the part stimulated and takes hold of the whole

exposed hemisphere (its dorsal, lateral, and medial surface). It

does not embrace merely field 29 d (Fig. 40).

(Legend to Fig. 40, textpage 87: Phenomenon of slow-spreading

depression. Spread of stimulating and discharging electrodes is

indicated in the scheme. Rabbit. A - prior to stimulation. L -

7 minutes after K. In each recording is indicated time from moment

of cessation of stimulation. Stimulation was made by induction

tetanization with a current applied through electrode S for a period

of 5 seconds (Leao, 1944).)

Rate of spread of depression equals 1-6 mm. per minute. About

5 minutes are required for the depression to spread from the frontal

to the occipital lobe. (Footnote: The spasmodic electrical activity

which sometimes arises in the regions seized by depression spreads

through the cortex at the same rate as the latter.) At each point it

develops with a more gradual character: 20-60 seconds are required

for a maximal depression of the electrical activity to develop. In

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the part stimulated the electrical activity can return to the normal

and the depression continues to spread through the cortex. The

second wave of depression can be provoked from the point stimulated

where the electrical activity in it has been reduced to a considerable

degree. A most stable depression is provoked at stimulation of the

frontal sphere of the cortex. At stimulation of the middle parts of

the cortex it spreads in both directions from the site of stimulation

and reaches frontal and occipital lobes simultaneously. At simultaneous

stimulation of the frontal and occipital lobe the waves of depression

proceed toward one another. At stimulation of the occipital lobe

spread of the depression often does not proceed through the cortex,

although depression that has arisen at stimulation of the frontal

lobe spreads to the occipital.

When depression of the "spontaneous" electrical activity seizes

a given sphere of the cortex, then there are oppressed or subside to

nothing: bioelectrical potentials which are provoked in this sphere

at stimulation of the nerves or of the receptors, bioelectrical

potentials which are aroused in a given area at electrical stimulation

of the opposite hemisphere, potentials which arise at direct electrical

stimulation of the cortical surface in a given area, strychninized

"spontaneous" spasmodic discharges which arise at local strychnine

poisoning of the cortex, and bioelectrical effects from the application

of eserine and acetylcholine (Leao, 1944).

Methods and conditions for obtaining the phenomenon of slow-spreading depression. As has already been said, this phenomenon is obtained at stimulation of the cortex by an induction current. The stimulation frequency should be above 30 per second. The phenomenon of depression can be obtained by using for stimulation right-angled shocks 6-12 milliseconds in length. Finally, this phenomenon can be obtained by applying to the cortex a constant current (0.1-4 milliamperes). According to the data of Leao and Morison (1945), the phenomenon of the slow-spreading depression arises on condition the cathode is applied to the cortex. However, Bures found that this phenomenon arises both under the action of the cathode and under the action of the anode (Bures, 1954a). In experiments on isolated strip of cortex (Burns, 1954) Leao's phenomenon arose both under the action of the cathode and under the action of the anode, although in the latter case a current of greater intensity was required. When in these experiments the anode was on the cortex, then the phenomenon of the spreading depression arose under a considerably greater current energy than the phenomenon of the rhythmical excitation of the cortical neurons (see textpp. 56-57); thus, not in all cases is Leao's conclusion correct, that the threshold of provocation of depression is lower than the threshold of provocation of the aftereffect in the form of intensified electrical activity.

The phenomenon of depression is readily obtained as a result of mechanical stimulations of the surface of the cortex. One or several

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contacts of a glass rod provoke it with a latent period of 10-40

seconds, and it then spreads through the cortex. This phenomenon

also arises as the result of unintentional, chance stimulations of

the cortex, connected with shift of the stimulating or discharging

electrodes (Sloan and Jasper, 1950). Verigo (1899) emphasized that

the cerebrum of frog is very sensitive to mechanical stimulations.

In his experiments light pressure of a glass rod on the brain was

sufficient for energetic deflection of the galvanometer: this part

became negative in regard to the adjacent ones. Even application of

the discharge electrode to the brain provoked the appearance of a

current. Below it will be seen that these observations of Verigo

offer interest in connection with the question under consideration.

Kaufman in curarized dogs observed that as a result of pressure on

the cerebral cortex with a glass rod a given part became negative

in regard to the other parts of the cortex (Kaufman, 1912). On the

other hand, even prior to the experiments of Leao it was known

that as a result of pressure rendered by application of an electrode

to the surface of a motor area of the cortex, the excitability of the

cortex is reduced, which is determined by the threshold of electrical

stimulation for provocation of a motor reaction (Markosian, 1941).

The phenomenon of the spreading depression can be caused by

chemical stimulations of the surface of the brain. KCl and $CaCl_2$

were found very effective in this respect. By application of a 1%

solution of KCl this phenomenon can repeatedly be provoked many times.

Physostigmine, acetylcholine, strychnine, cocaine, histamine, and

adrenalin do not provoke the phenomenon of spreading depression at

their local application to the cortex (Leao, 1944). Sloan and Jasper

(1950) were able to obtain /with application of a concentrated solution
                          it

of strychnine to the cortex.

The phenomenon of the slow-spreading depression is obtained in

rabbits with the greatest constancy. Leao readily obtained it in

pigeons, and Bures obtained it in mice and rats. It is far more

difficult to obtain it in cats and monkeys. There is indication

that it can also arise in cerebral cortex of man at the time of

cerebral operations (McCulloch, 1949).

The phenomenon of the slow-spreading depression is obtained in

narcotized animals, the depth of the narcosis having no particular

significance (Marshall, 1950). It is obtained in lightly curarized

animals (Whieldon and Harreveld, 1950). It is obtained on non-

narcotized animals rendered motionless by intersection of the spinal

cord at the boundary with the medulla oblongata (Sloan and Jasper,

1950), and finally in non-narcotized animals with the central nervous

system intact (Bures, 1954b). Gedevani (1947, 1949) came to the

conclusion that the phenomenon of the slow-spreading depression of

the cortical electrical activity occurs only during deep barbituric

narcosis. The data just cited on the possibility of obtaining the

phenomenon considered in non-narcotized animals place this conclusion

in doubt. Moreover, it has been ascertained that a rate of spread of

depression equal to 1-6 mm. per minute is not markedly altered,

depending on the depth of the narcosis, and it is the same in the

absence of narcosis (Sloan and Jasper, 1950; Bures, 1954b). On the

other hand, for provocation of depression during narcosis the same

intensity of constant current is required as in the absence of

narcosis (Bures, 1954b).

Recently it was ascertained that under the effect of larger

doses of pentobarbital the threshold of provocation of the slow-

spreading depression phenomenon is somewhat increased and the rate

of its spread is somewhat reduced (Whieldon and Harreveld, 1951).

However, even with such doses of the narcotic as that at which the

"spontaneous" electrical activity of the cortex dwindles almost

to nothing and at which not even a knee reflex is provoked, it is

possible to cause the phenomenon of the slow-spreading depression.

It evidently ceases to arise during deep ether narcosis that brings

breathing to a standstill (Harreveld and Stamm, 1953a).

At the spreading of the depression strips of cortex 5-8 mm.

in width that are seized by it, i.e. the preceding part, begins to

return to normal when the depression in the latter part reaches its

maximum. In connection with this, this phenomenon is not observed
or is expressed worse when the interpolar distance of the discharge
electrodes is greater (more than 3-4 mm.), since thereby one of the
discharge electrodes can be in a region seized by a wave of depression
and the second can be in a normal part of the cortex or in a part
where the depression is already attenuating. The phenomenon of
depression of the "spontaneous" electrical activity cannot be recorded
because of the layer of the conducting fluid which covers a relatively
large area of the cortex. In this case the part under the discharge
electrode can be found in a state of depression, but it will be masked
by the bioelectrical activity of the adjacent parts of the cortex
connected with the electrode through the conducting fluid (Harreveld
and Stamm, 1951).

A number of other factors determining the readiness and the
possibility in general of obtaining the phenomenon of the spreading
depression will be considered below.

Concerning the spread of depression. Depression is not spread
through a cocainized strip of cortex (Leao, 1944). If surface section
of the cortex is effected, then the depression likewise is not spread
through this side of the section. Thermocoagulation of the cerebral
membrane and of the two upper layers prevents the spread of the depression
through the injured area. Thus, for spread of depression the surface

cortical structures are necessary. Evidently provocation of the

phenomenon of the slow-spreading depression is connected with the

activation of the surface structures. The results of the following

experiment are indicative of this: the stimulating electrodes

that diverge 0.5 mm. from one another along the verticals were

sunk deep into the cortex, and threshold intensity of current was

sought for provocation of the Leao phenomenon. The least intensity

of current was needed when the upper electrode was on the surface of

the cortex. When the electrodes were in the white matter, then a

3-4 times greater intensity of stimulation was required for this

than at stimulation of the surface of the cortex (Leao and Morison,

1945). After separation (by knife) of the upper layers of the cortex

from the deep ones, the spread of depression did not cease. Hence,

it is possible to conclude that the long, associative cortical

pathways in the white matter under the cortex are not necessary for

spread of the depression. Section of the thalamus-cortical connections,

as well as infringement of all cortical connections with subcortical

formations shows no effect on the spread of the depression. From

the "undercut" part of the cortex, at its stimulation, the depression

spreads over the whole cortex (Sloan and Jasper, 1950). Finally, the

phenomenon of the slow-spreading depression was observed at stimulation

of an isolated strip of cortex (Burns, 1954).

Thus, the slow-spreading depression of the electrical activity
is a cortical phenomenon.

The first hypothesis which can arise is the formation and
spread of a depression connected with the activity of the cortical
neurons, with their excitation that spreads through the usual well-
known principle, i.e. from one neuron through the synaptic endings
of its axon to the following neuron, etc. However, the following
series of facts contradict this hypothesis.

1) The preceding parts of the chapter in question have been
devoted to an analysis of the biopotentials arising at stimulation
of the surface of the cortex, to a clarification of the mechanism
of their spread, and have concerned, as was indicated, bioelectrical
phenomena linked with the activity of the neurons and of neuronic
elements of the cortex. These bioelectrical phenomena were very altered,
depending on the depth of the narcosis, and the compass and rate of
spread of the biopotentials in particular were changed. We see too
that narcosis shows no appreciable effect on slow-spreading depression.

2) The extremely small rate of spread of depression, equaling on
the average 3 mm. per minute*, can be explained from the point of view
of facts known concerning the mechanism of transmission of excitation
from neuron to neuron. (*That is, 2000 times less than the rate of
spread of the excitation through the fibers of layer I of the cortex.)

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3) The process of the transmission of excitation from neuron
to neuron is very responsive to anemia; even "spontaneous" electrical
activity of the cortical neurons disappears 10-12 seconds after
cessation of circulation of blood in the brain. (Footnote: According
to the data of Ten-Cate and Horsten (1954), after 15-20 seconds.)
If at the time of the slow-spreading depression anemia of the brain
is caused by pressure applied to the carotid and vertebral arteries,
then after return of the "spontaneous" electrical activity to normal
it is shown that the depression continued to spread even at time of
anemia.

4) Sloan and Jasper found that if part of the cortex is isolated
by deep circular section from the rest of the cortex, then the depression
caused by stimulation of any area of the cortex spreads also to the
isolated part. It ceases to spread in it if the edges of the section
are drawn 1.5-2 mm. apart. Thus, for spread of depression through
the cortex physical contact of its parts is sufficient. Thus, spread
of depression of cortical activity through the cortex is not connected
with spread of excitation through the cortical neurons. Consequently,
identification of the Leao phenomenon and of the cortical inhibition
process (Gedevani, 1947, 1949) cannot be recognized as correct.

On the other hand, there is a series of facts which indicates
that the cortical neurons can participate in the arising of the Leao

phenomenon.

1) As said above, the depression continues to spread through the cortex at the time of 10-12-second anemia, but at the time of anemia it is impossible to provoke by stimulation of the cortex the phenomenon of the slow-spreading depression. On this basis the hypothesis was formed, that depression arises in consequence of the excitation of neurons around the stimulating electrodes, but its further spread does not depend on the transmission of excitation from neuron to neuron. It should be noted that not in one of the investigations of this phenomenon has it been found that it occurs at the time of stimulation; in all the recordings at the time of stimulation the tubes were shut off and the electrical activity was recorded at cessation of stimulation. At the same time clarification of this, whether excitation of the neuronic elements of the cortex occurs and what elements are implicated in the reaction, is fully possible, as we have seen, a thing that might have significance for understanding the phenomenon of the slow-spreading depression.

2) In rabbits it is possible to provoke the phenomenon of the slow-spreading depression in the opposite hemisphere (Leao, 1944). For this it is necessary to use a more intense stimulation than for provocation of it in the hemisphere stimulated. Though the latent period of the setting in of the depression in the opposite hemisphere

is greater than in the one stimulated, still it is considerably less
than the latent period of the setting in of it in the part of the
hemisphere stimulated, found at the same distance from the point
stimulated as the part of the opposite hemisphere symmetrical to this
point. It is easier to provoke depression in the opposite hemisphere
by stimulating those parts in which there is the greatest number of
anatomical connections with the opposite symmetrical part. Depression,
if it arises in the opposite hemisphere, forms in a part symmetrical
with that stimulated; hence, it spreads to the entire opposite
hemisphere.

Cross depression is the result of excitation of the fibers of
the corpus callosum. After section of the corpus callosum stimulation
ceases to provoke the phenomenon of depression in the opposite
hemisphere. Cross depression is not a consequence of the process
itself of depression in the part stimulated. This conclusion issues,
first of all, from the fact that at time of depression caused in one
hemisphere and spreading slowly through it no appreciable changes
occur of the electrical activity of the cortex of the opposite
hemisphere (Bures, 1954a). The accuracy of this conclusion is
demonstrated by the results of the following experiment: in the
frontal lobe the cortex was removed and stimulating electrodes were
placed in the white matter: as a result of stimulation, at a

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symmetrical point of the opposite hemisphere a depression arose and
spread over the entire opposite hemisphere; on the side of stimulation
the phenomenon of the slow-spreading depression did not arise.

3) As said, spread of depression in the hemisphere stimulated
can occur without the participation of the long association pathways.
However, there are facts which indicate that in certain cases the
phenomenon of the depression can develop with the participation of
the long cortico-cortical pathways - the depression "skips" from
the stimulated part to a remote part, i.e. primarily it develops with
a short latent period in a remote part and just from there is spread
over the whole cortex (Marshall, 1950).

The series of facts cited testifies in favor of the phenomenon
of the slow-spreading depression being able to arise under the influence
of excitation impulses that come to the cortex. Still this extremely
important question has not until now undergone special study. No
attempt has been made to clarify whether the phenomenon of the slow-
spreading depression arises at stimulation of the nerves and receptors,
i.e. under the influence of the afferent impulses that arrive at one
or another cortical analysor. However, since there are no indications
of this estimate, though there is a very great number of works on the
study of the effect of peripheral stimulations on the electrical activity
of the cortex, then it is possible to think that Leao's phenomenon does

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not arise at peripheral stimulations. Apparently it does not arise

even during stimulation of the meshlike formation of the stem of the

brain, as can be concluded from examination of the records of Arduini

and Lairy-Bounes (1952).

At the very beginning it was said that the cortical biopotentials

that arise with various methods of cortical stimulation attenuate or

dwindle to nothing. The attenuation or disappearance of the effects of

direct electrical stimulation of the cortical surface must indicate the

depression of the top dendrites of the pyramidal neurons and/or of the

fibers of layer I. Attenuation or disappearance of effects of electrical

stimulation of the opposite hemisphere must indicate depression of the

elements of layer III, because mainly the callosal fibers terminate in

this layer. The attenuation and disappearance of the bioelectrical

effects from stimulation of the sensory nerves and receptors (Leao,

1944; Marshall, 1950) at the time of the depression that takes hold

of the area of the corresponding analysor in the cortex indicates

depression of neuronic elements mainly of layer IV of the cortex, in

which chiefly the afferent fibers terminate. As said, "spontaneous"

electrical activity does not disappear but only attenuates somewhat

after thermocoagulation of the upper layers. Hence, the fact that

"spontaneous" electrical activity at the time of spreading depression

sharply attenuates or dwindles to nothing testifies to a depression

of the elements of all layers of the cortex, V and VI in this number.

Certain facts obtained by Sloan and Jasper (1950) indicate,
as it were, that when a depression wave seizes hold of a motor area
of the cortex excitation of the Betz cells in the deep layers of the
cortex is reduced. The threshold of provocation of movement by
electrical stimulation of the motor area of the cortex was considerably
heightened (from 7.2 to 9.2 v.) when the depression wave seized this
area, i.e. when the "spontaneous" electrical activity here abruptly
weakened. When the depression wave reached the motor area of the
cortex, from which persistent motor effects were provoked during
stimulation at a rhythm of 1 per second, then these movements ceased
being aroused; they began to be provoked again when the electrical
activity returned to normal.

Finally, it is possible to use the results of but one experiment
for proof that slow-spreading depression takes hold of the association
neurons of the deep layers of the cortex. During stimulation of the
cortex, for instance, in the anterior part of the hemisphere at a
symmetrical point of the opposite hemisphere characteristic bioelectrical
effects arise, stipulated by the impulses of excitation that proceed
through the fibers of the corpus callosum, as already stated repeatedly.
When the slow-spreading depression provoked by stimulation through a
second pair of stimulating electrodes placed on the surface of the
occipital region of the cortex reaches the region of the location of
the first stimulating pair, then the stimulation at this time of a given

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region stops provoking bioelectrical effects in the opposite hemisphere
(Leao, 1944). According to neurological data, the main source of origin
of the callosal fibers in layer VI of the cortex: the callosal fibers
are axons of the cells chiefly of layer VI of the cortex (Chang, 1953).
Consequently, the slow-spreading depression takes hold of these association
neurons too.

Changes in the nuclei of the thalamus at the time of the slow-
spreading depression through the cortex. Investigation of the electrical
activity of the thalamus at the time of the slow-spreading depression
in the cortex, which was carried out by Winokur and co-workers (1950),
gave no clear facts. It was noted that when in the cortex at the time
of the spreading depression individual slow fluctuations of the biopotential
arose they were registered also in the thalamus.

Important facts on this question were obtained by Sloan and Jasper
(1950) who found that at the time of the depression that seizes a given
cortical field the electrical activity of the thalamic nucleus functionally
linked with this field attenuates. It was demonstrated that depression
of the electrical activity in the thalamic nucleus is a secondary
phenomenon and the attenuation of the cortical activity in the corresponding
field of the cortex is primary. The experiments on the basis of which
these conclusions were made have consisted of the following. During
stimulation of the cortex depression took hold of the entire cortex.

When it spread to the portion of the cortex anterior to the sulc.
cruciatus, then depression of the electrical activity was registered
also in the nucl. ventr. ant. thal. After isolation of a given
cortical field from the rest of the cortex (circular section) the
depression spread over the entire cortex, apart from the isolated
field. The electrical activity in the thalmic nucleus was no longer
altered.

Long nonfluctuating potentials at the time of the slow-spreading
depression. When a slow-spreading depression provoked in any manner
reaches the spheres of the cortex where the nonpolarizable discharge
electrode connected with the galvanometer[*] is established, then simul-
taneously with depression of the "spontaneous" electrical activity a
long potential of several minutes' duration is registered. ([*]In Leao's
experiments a second electrode was located on bone; in the experiments
of Bures, on the surface of the other hemisphere.) This potential has
2 phases: at first a negative potential arises (7-15 milliv. according
to Leao, 1947, and Bures, 1954b; 36 milliv. according to Harreveld and
Stamm, 1951), which lasts 0.7-2 min., reaching a maximum after 0.5-1
min.; then this point of the cortex becomes positive, the positive
potential has less amplitude and lasts 2-5 min. (Fig. 41, textpage 94:
Long nonfluctuating potential registered at the time of a slow-spreading
depression. The potential is recorded by a galvanometer from a point

of the cortex 4 mm. distant from the stimulating electrodes on

the cortex. Horizontal line - period of stimulation; vertical line

= 1 milliv. Rising of curve - negativity. The dot indicates the

beginning of depression of the electrical activity in the corticogram

(which is not presented). Time marks 10-second intervals (Harreveld

and Stamm, 1951).

This potential spreads from the locality of stimulation to

the whole side, arising in each given locale of the cortex side by

side with alteration of the "spontaneous" electrical activity and

excitability (Leao, 1947). Thus, the latent period of the arising

of a prolonged potential corresponds to the latent period of the

arising of the depression of the "spontaneous" electrical activity.

According to Harreveld and Stamm's data (1951), the potential

arises 10-40 seconds earlier than the start of depression of the

electrical activity. Parallel with the growth of the negative

potential a progressive attenuation occurs of the "spontaneous"

electrical activity; the latter begins to be reduced at the end

of the positive phase of the slow potential.

The long potential being considered does not arise simultaneously

for the various levels of the cortex. This was established in experiments

with simultaneous discharge of potentials from the surface of the cortex

and from the middle layers of the cortex, from the surface and from the

deep layers of the cortex (Leao, 1951). It has been shown that the

negative potential arises in the middle layers later than in the

surface ones, in the deep ones later than in the middle ones, and in

the white matter it does not arise. The rate of spread of negativity

from the cortical surface into the depths is of the same order as the

rate of its spread longitudinally through the cortex. Thus, from

the point of the cortex stimulated the negative potential spreads,

on the one hand, deep into the cortex and, on the other hand, along

the cortex. In each part where a wave of depression occurs the

negative potential arises at first in the surface layers of the

cortex and spreads, on the one hand, deep into the cortex of the

part in question and, on the other hand, to adjacent surface elements

of the cortex (Leao, 1951).

Thus, the depression which corresponds to the long nonfluctuating

potential spreads from the surface of the cortex to layer VI at the

rate of 1-6 mm. per minute. Hence, it follows that reduction of the

excitability of the cortical neurons of the different layers must

occur at a different time. However, special experiments for clarification

of this question have not been made.

In the depths of the cortex a positive phase indicating that

the surface parts of the neurons (top dendrites of the pyramid neurons)

are negative in respect to their deep parts (to the bodies and basal

dendrites of the pyramidal neurons) precedes the arising of a long

negative potential. On this basis Leao concludes that the negative

potential travels from the surface of the cortex through the nerve

elements perpendicularly oriented to the surface, i.e. downwards

through the top dendrites to the bodies of the pyramids. However,

no experiments whatever have been carried out to clarify this

question.

The long nonfluctuating potential arises in the cortex as

the result of prolonged cortical anemia caused by compressing the

blood vessels. Anemia of the brain after 2.5-5 minutes leads to the

development of steadfast electronegativity of the cortex in regard

to the indifferent electrode. The negative potential reaches some-

what greater magnitudes than at the time of slow-spreading depression.

The cortex remains negative in regard to the indifferent electrode

so long as the vessels are compressed; the negative potential

disappears several minutes after restoration of blood circulation.

The negativity of the cortex that arises as the result of the com-

pression of the blood vessels does not develop simultaneously in all

parts of the cortex: usually one part of a hemisphere becomes

negative and then, after 1-2 minutes, negativity of other parts of

the cortex too develops (Leao, 1947). Kaufman (1912) observed

entirely similar phenomena during asphyxiation provoked by cessation

of artificial respiration. As the result of asphyxiation the cortex

became electronegative and the difference of the potentials of the

optical and motor areas of the cortex was increased. On the basis

of the illustrations of it, it can be concluded that the optical

area of the cortex became electronegative in regard to the motor.

Kaufman apparently made an entirely accurate conclusion, that the

cells of the different areas of the cortex possess a different

susceptibility to CO_2, which is the reason too for the phenomenon

described above. Recently it was ascertained that at inhalation

of 35% CO_2 by the animal "spontaneous" electrical activity of the

optical area of the cortex ceases considerably earlier than the

"spontaneous" electrical activity of the auditory and motor areas

(Gellhorn, 1953). Evidently this circumstance too stipulates a

different rate of development of the negativity of the various

parts of the cortex during anemia. Pavlov at analyzing the

pathophysiological mechanism of catatonia came to the important

conclusion that the motor elements of the cortex "have nothing in

common in a structural or chemical respect, or probably both in

the one and in the other, and therefore pertain in an identical

way to the source generating the symptoms of pain, thus differing

from the other elements of the cortex: optical, auditory, etc."

(Pavlov, 1919). There are data that testify to the selective

effect of mescaline on the cells of the optic area. Under the

effect of mescaline sharp infringements occur of the conditioned-

reflex activity, which are localized within the limits of the optic

analysor, whereas changes in the cutaneous and auditory analysors

are insignificant (Alexandrovskii, Babskii and Kriazhev, 1936).

It should be noted that, according to Bekhterev (1896), the different

areas of the cortex develop at far from an identical time.

Thus, the fact that the different parts of the cortex develop

at a different time the negative potential during anemia receives a

satisfactory explanation if it proceeds from the position of the

different properties of the neurons of the different areas of the

cortex. From this point of view it is possible to explain too the

development at a different time of the negativity of the different

layers of a given portion of the cortex during anemia. Leao found

that at anemia the surface layers of the cortex first become

negative and the deep layers of the cortex develop the negative

potential later. Moreover, approximately the same temporal relation-

ships are observed as at the time of slow-spreading depression (Leao,

1951).

There are histological data indicative that the elements of

the surface layers of the cortex, the cells of which develop later

than the cells of the deep layers (Bekhterev, 1896), and which in

the opinion of a number of neurologists are phylogenetically most

recent (Sarkisov, 1948, 1952; Sarkisov and Poliakov, 1949), possess

greater sensitivity to different influences than the elements of

the deep layers of the cortex. For instance, during the schizophrenic

process pathomorphological changes are discovered mainly in the elements

of the surface layers of the cortex (Zurabashvili, 1950). Thus, the

fact that first during anemia the surface layers of the cortex develop

electronegativity receives satisfactory explanation if it issues from

the position that the greater the sensitivity the greater the susceptibility

to injury of the surface neuronic elements as compared to the deep, i.e.

if it issues from the fact that parabiosis of the surface elements

develops earlier and more quickly. On the basis of the facts cited

above it is possible to conclude that the top dendrites of the pyramid

neurons are more sensitive to anemia than the body of these neurons.

This conclusion agrees with certain data of histological investigations

indicative of the greater sensitivity of the dendrites to different

influences. For example, it has been established that protoplasmatic

offshoots react first to the injurious effect of an electrical current;

in the system of dendrites severe degenerative changes are discerned

when the cell bodies do not yet disclose noticeable pathomorphological

changes (Zurabashvili and Cholokashvili, 1948).

Compression of blood vessels for 1 minute causes complete

cessation of the "spontaneous" electrical activity of the cortex,

but negativity of the cortex does not thereby develop. Nevertheless,

one-minute anemia shows a marked effect on the potential of a given

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part of the cortex if it is provoked at a time when the part of the

cortex in question is seized by a wave of slow-spreading depression:

the anemia intensifies the slow negative potential, and this change

remains for several seconds after restoration of the blood circulation

(Leao, 1947). Thus, the cortical fields seized by slow-spreading

depression show themselves with sensitivity to anemia boosted.

According to Leao, this fact indicates that at the time of slow-

spreading depression in the cortex changes arise analogous to those

which are provoked as a result of cessation of the circulation of the

blood. (Footnote: On the other hand, speaking in favor of this is

the fact that prolonged compression of the blood vessels on the

background of a slow potential caused by stimulation of the cortex

cannot produce in a given sphere a new slow potential, i.e. the

cortex in this respect is just like a refractor (Harreveld and Stamm,

1953b).) This conclusion has been logically substantiated, since

at the time of slow-spreading depression the phenomena of infringe-

ment of the circulation of the blood are observed.

Pial circulation and the phenomenon of the slow-spreading

depression. According to the data of Leao (1944a) at the place of

the stimulation of the cortex when depression of the "spontaneous"

electrical activity develops dilation of the arteries and veins sets

in: tiny blood vessels, hitherto invisible, become visible. This

change of the pial vessels spreads slowly in every direction from

the site of stimulation parallel with the spread of the slow potential

and of depression of the "spontaneous" electrical activity. In each
part of the cortex maximal dilation of the blood vessels sets in
0.5-1.5 min. after it begins to be noted, i.e. approximately when
the negative potential reaches a maximum. At the time of maximal
dilation the diameter of the blood vessels of the pia mater is
increased 50-100%. At this time the general blood pressure, measured
at the carotid artery, is not changed. The lumen of the blood vessels
returns to normal 1.5-3 minutes after its maximal dilation (Leao,
1944a).

If at the time of the depression electrical activity of a
convulsive character (see above) appears, then changes of the
circulation at the time of this abnormal electrical activity is
the same as at the time of pure depression. (Footnote: in connection
with this it should be noted that in epileptic patients intensification
was observed of the blood current in the cortical region seized by
convulsive discharges (Penfield and coworkers, 1939). The convulsive
activity observed sometimes during spreading depression is similar to
that which arises during experimental epilepsy (Rosenblueth and Cannon,
1942). On this basis, as well as on the basis of certain other facts,
it is assumed that the phenomena of the spreading depression and of
convulsive spreading activity are closely linked with one another (Leao,
1944).)

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Thus, as the result of several seconds of tetanic stimulation of the cortical surface a vasodilation wave arises which in rabbit takes hold of the whole cortex for several minutes (with the exception of field 29 d).

During a certain intensification of stimulation, as already said, depression develops too in the opposite hemisphere; furthermore, dilation of the blood vessels arises there, being at first in a part symmetrical with the stimulated area, then all 3 reaction components, the depression of the "spontaneous" electrical activity, the slow nonfluctuating potential, and the vasodilation, spread over the entire opposite hemisphere with the rapidity well known to us.

Leao's observations on changes of blood circulation in the pial vessels is extremely hard to harmonize with the changes of bioelectrical potentials of the cortex that are observed at this time. Why should dilation of the blood vessels accompany depression of "spontaneous" electrical activity and the development of the electronegativity of a given part of the cortex when these bioelectrical phenomena are characteristic to a state of anemia of the cortex?

With very recent investigations (Harreveld and Stamm, 1952) a series of facts not agreeing with Leao's observations were obtained. First, it was ascertained that at the time of slow-spreading depression change occurs of the volume of the cortex: the volume of a given part

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of the cortex begins to lessen when a prolonged nonfluctuating
potential develops, accompanying the depression. Hence, the
conclusion was made that the slow-spreading depression is attended
by a wave of vasoconstriction. Secondly, it was ascertained that
at this time a drop of oxygen pressure occurs in the cortex. Hence,
the hypothesis was made that spreading depression of cortical
activity is an asphyxial phenomenon provoked by a wave of vasoconstriction.
Thus, it is shown that the galvanometric experiments of Kaufman (1912)
with a study of the bioelectrical phenomena in the cortex during asphyxia
have a direct relationship to the phenomenon being considered.

Harreveld and Stamm did not observe dilatation of the pial blood
vessels during spreading depression. According to their data, dilatation
of these vessels sets in after the wave of vasoconstriction seizes a
given part of the cortex. They think that this dilatation of the pial
vessels sets in under the effect of metabolites cumulative in the cortex
during the period of the vasoconstriction.

Is the slow-spreading depression a normal physiological
phenomenon or an abnormal reaction of the cortex? This question arises
in connection with the researches of Marshall and coworkers (Marshall,
1950; Marshall, Essig, and Dubnoff, 1951), according to which one of
the main conditions for the arising of the phenomenon of the slow-
spreading depression is desiccation of the exposed cortex.

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The arising of Leao's phenomenon can be prevented by covering
the cortex with a layer of mineral oil. However, Leao's phenomenon
can be obtained too in this case, i.e. after the cortex is protected
from drying if dehydration of the brain is caused by introduction of
12-30 ml. of a 90% solution of glucose into the blood; thereby
reduction occurs of the volume of the brain and disappearance of
the cerebrospinal fluid under the arachnoid membrane.

The arising of the Leao phenomenon is linked with injury of
the pia-arachnoid system of the exposed cortex and with change of
the normal physico-chemical processes in this system. Thus, slow-
spreading depression is considered a reaction of injured cortex, a
reaction not arising in normal cortex and having no relationship
to its normal physiological activity.

The following series of facts, however, contradicts this
conclusion. The phenomenon of the slow-spreading depression can
be produced several minutes after removal of the dura mater, i.e.
drying of the cortex as the result of its long contact with the
surrounding air is not a requisite condition for provocation of
the phenomenon of depression (Sloan and Jasper, 1950). Depression
spreads through the regions of the cortex covered with intact
membranes and cranium (Harreveld and Stamm, 1951), which has been
demonstrated in the following way: two small trepanation openings

were made in the skull, one in the frontal, the other in the occipital

region. At stimulation of one of these regions, in the other after

several minutes a typical phenomenon of depression of the "spontaneous"

electrical activity and a corresponding prolonged potential developed.

The rate of spread of depression in these conditions equaled 2-3 mm.

per minute (Harreveld and Stamm, 1951). Thus, depression spreads

through the unbared cortex, and the rate of its spread thereby is

that which is ordinary for this phenomenon.

Harreveld and Stamm think that the phenomenon of slow-spreading

depression is easier to observe in rabbits than in cats and monkeys

in connection with this, that the cerebral cortex of cat and monkey

has deep sulci, the larger blood vessels are covered by the thick

pia mater, and all these circumstances render registration of the

phenomenon difficult.

After the experiments of Bures (1954b) made on non-narcotized

animals in which the cortical potentials were registered without

penetration into the cavity of the skull, it is possible to consider

established that the phenomenon of the slow-spreading depression can

arise in normal cerebral cortex and that this is a physiological

phenomenon.

It might be possible to assume that the depression of the

"spontaneous" electrical activity registered at the time of the Leao

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phenomenon, as well as of the bioelectrical reactions of the cortex

expressed by direct stimulation or by stimulation of the receptors,

depends on a worsening of the discharge conditions of the potentials

from the surface of the cortex in connection with change of the blood

volume of the vessels of the pia mater. However, in the first place,

at simultaneous discharge of bioelectrical potentials from the cortex

and from the nucleus of the thalamus connected with this part of the

cortex it is discovered, as already said, that at depression of the

electrical activity in the cortex the electrical activity of the

corresponding subcortical nucleus attenuates. This indicates that

the depression of electrical activity registered from the surface

of the cortex reflects the true aspect of things. In the second

place, when depression seizes the optical area of the cortex, then

the bioelectrical reactions ("primary responses") of the optical cortex

attenuate at stimulation of the optic nerve, but the initial quick

potential expressing excitation of the endings of the afferent fibers

in layer IV of the cortex does not thereby attenuate (Marshall, 1950).

This fact indicates that attenuation of cortical bioelectrical potentials

arising in response to peripheral stimulations is not a consequence

of the change of conditions of discharge but reflects an oppression,

the depression of the corresponding cortical neurons.

Certain considerations regarding the origin of the phenomenon

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of the slow-spreading depression. On the basis of the facts cited
above it is possible to think that at the base of the phenomenon being
considered lies a vascular reaction. Evidently as a result of
different kind of stimulations applied to the pail surface constriction
arises of the vessels of the pia matter and of the inner vessels of the
cortex, which leads to temporary asphyxia of the neuronic elements of
a given part of the cortex. The last circumstance stipulates, on the
one hand, its electronegativity and, on the other hand, attenuation
of "spontaneous" electrical activity and attenuation of the bioelectrical
potentials arising with different methods of stimulation of the cortex.
The mechanism of the arising of this vascular reaction and the mechanism
of its spread have not been clarified at the present time.

According to data to be had (see Klosovskii, 1951) on arteries
and veins of the pia mater, down to those smallest in caliber, there
is a great number of nerve fibers passing along the shaft of the
vessel and encircling it in the form of a spiral. Usually these
fibers proceed in the form of tufts consisting in part of medullated,
in part of unmedullated fibers. Some of these fibers are, as thought,
vasculomotor, since their endings branch out among the smooth muscle
fibers of the vessels. Other fibers must be sensory, connected with
the receptor end apparatuses which are found in the adventitia of
the vessels of the pia mater. The nerve plexus proper of the pia
mater is formed by numerous unmedullated nerve fibers.

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The nerve fibers of the vessels of the brain are a continuation
of the nerve plexus of the pia mater and enter into the brain together
with the vessels. The diameter of the intracerebral vascular nerve
fibers is very small (0.5-1 micron, maximum 2.5 microns). They are
devoid of a myelin membrane. They may be a continuation not only of
the unmedullated fibers of the plexus of the pia mater but also a
continuation of the thick medullated fibers that lose their myelin
at entering into the brain matter and that break up into delicate
fibers.

The nerve fibers of the vessels of the brain and of the pia
mater are of sympathetic and parasympathetic origin. In Klosovskii's
opinion (1952) the sympathetic portion of the nervous system can
participate in redistribution of the blood in the brain, constricting
and even closing the cerebral capillaries between the separate
arterial branches.

Certainly at electrical, mechanical, or chemical stimulation
of the pial surface of the cerebral cortex not only the neuronic
elements of layer I of the cortex but also nerve plexi of sympathetic
and parasympathetic origin located in the pia mater and along the
course of its vessels should be stimulated primarily. It is possible
to assume that the phenomenon of the slow-spreading depression,
discovered by Leao, is stipulated by stimulation of the nerve plexi

of the pia mater and thus to explain the arising of vascular reaction

in the region of stimulation of the cortex[*], but, all the same, the

mechanism of the spread of this phenomenon through the cortex remains

obscure. Does the formation of some metabolites which, in their turn,

cause vascular reaction of the horizontal part of the cortex lying

in line perhaps govern a derangement of the circulation of blood in

a given part of the cortex and reduction of oxygen pressure? Does

excretion of some chemical substances in the surface layers of the

cortex which, acting in a stimulating way on the nerve plexi of the

pia mater, cause vascular reaction perhaps also occur during excitation

of the cortical neurons under the effect of impulses of excitation

(for instance, from the callosal fibers)? ([*]As Kirzon (1934)

established for excitation of the sympathetic system, stimulation

of it by right-angled impulses at a frequency of up to 70 per second

is most effective. It is characteristic that the phenomenon of the

slow-spreading depression is provoked more easily if the cortex is

stimulated by right-angled impulses of greater length. For instance,

in many of Sloan and Jasper's experiments (1950) the phenomenon of

the slow-spreading depression arose after 2 seconds of stimulation

by right-angled impulses 12.8 milliseconds in length (8 volts, 40

per second). In Harreveld and Stamm's experiments (1953b) during

cortical asphyxia its faradic stimulation provoked no slow potential

(component of Leao's phenomenon), but this potential could be provoked
at stimulation of the cortex by right-angled impulses.) It has been
established that at tetanic stimulation of the nerve fiber excretion
of some substance from it out through the Schmidt-Lantermann's incisures
occurs, this process having a greater latent period (Kuparadze, 1953,
1954). As we saw, the fibers of layer I have as the main source of
their origin the ascending axons and collaterals of the neurons of
all subjacent layers of cortex, and the natural excitation of the
fibers of layer I assumes a preliminary excitation of the neurons
of the other layers of the cortex. It is possible to assume that
during long, intensive excitation of the cortical neurons elimination
occurs of a considerable amount of physiologically active substances
from the excited fibers of layer I into the tissue fluid, excitation
of the nerve plexi of the pia mater, vascular reaction, and, stipulated
by it, the phenomenon of the slow-spreading depression. Finally, very
little of the hypothesis just expressed has factual basis. For
instance, Leao's researches (1944) and recently those of Harreveld
and Stamm (1953c) have revealed that neither yohimbine and ergotamine
nor atropine show an effect on the phenomenon of spreading depression.
Hence, it was concluded that the wave of vasoconstriction that lies,
as needs to be thought, at the base of this phenomenon is not stipulated
by adrenalin-similar or acetylcholine-similar substances. A circumstance
that makes the question even more involved is that during certain

conditions, as already said, depression of electrical activity

can give place to convulsive electrical activity, the latent period

of the arising of which and the rate of spread through the cortex

are the same; moreover, the slow electrical potential also arises.

The passing over of depression of electrical activity into convulsive

electrical activity is observed at repeated stimulations of the cortex

that cause at first the phenomenon of depression during considerable

rise of the general blood pressure, during local poisoning with a

5-10% solution of acetylcholine and, finally, during the effect of

CO_2 (inhalation of air containing 7-15% CO_2). They think that the

arising of convulsive electrical activity in all these cases is

connected with attenuation of vasoconstriction. For instance, it

is known that CO_2 shows a powerful vasodilatation effect on the blood

vessels of the brain.

The solving of the question of the nature of the phenomenon being

considered is not facilitated by drawing on a series of well-known data

on alteration of the circulation of the brain and of its electrical

activity during a series of physiological and pathological conditions.

For instance, during natural sleep the blood vessels of the brain are

constricted. On the contrary, at time of stimulation of the sense

organs dilatation of the blood vessels occurs in certain regions of

the cortex: at time of vestibular stimulation - in the motor area

of the cortex; at time of illumination of the eye - in the optical

region of the cortex; at this time in other regions of the cortex a

certain constriction of the blood vessels is observed (Klosovskii,

1951, 1952). On the other hand, with the vasoconstriction of the

blood vessels of the brain is linked a tendency to increase of

amplitude of the slow fluctuations and attenuation of the quick ones;

with vasodilatation, on the contrary, is linked a tendency to

intensification of the quick fluctuations and attenuatimn of the

slow (Darrow, 1946).

Finally, if Bykov's point of view is considered (1952, 1955),

then the vascular reaction arising during excitation of the cortical

neurons under the influence of the callosal impulses can be treated

as an expression of this principle, that the nerve impulses by causing

a change of state of certain cortical neurons simultaneously cause

change too in the apparatus of supplying the same group of neurons.

According to the hypothesis which Bykov proposed, in the process of

reflex activity nerve influences can arise on the trophism of the very

nerve formations themselves. The mechanism of this phenomenon is

presented in the following way: the axons of a given group of cells

not only form synapses to other neurons, but their ramifications can

produce terminals to blood vessels in the nerve networks and possibly

too in the intercellular matter. Because of this, the nerve impulses

that arise in one group of nerve cells not only stimulate other neurons
but simultaneously influence the entire trophism of these neurons. This
hypothesis receives morphological basis, since it is shown that the
endings of the fibers of the afferent and efferent systems of the
cerebral cortex are distributed in the cerebral cortex not only to
the neurons but also to their surrounding vessels (Dolgo-Saburov, 1953).

Thus, the occurrence of the phenomenon of slow-spreading depression
remains obscure. The biological importance of this phenomenon is not
known likewise, since the experiments were not made for the purpose
of studying changes of behavior and of conditioned reflexes at the
time of the phenomenon of the slow-spreading depression.

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Chapter III

Oscillographic Investigation of Foci of Heightened Excitability in the Cerebral Cortex

We now run into complex bioelectrical phenomena, where the
effect of cortical stimulation is determined not only and not so much
by the anatomy of the formations being stimulated and by the character
of the stimulation, but mainly by those physiological reactions which
proceed in the cortex at the moment of stimulation; we run into
phenomena where a "given cortical 'focus' loses the importance of an
apparatus with any one functional purpose" (Ukhtomskii, 1911); and

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we run into phenomena that evidently lie at the base of special

cortical mechanisms and which have not been investigated electro-

graphically up to now.

Numerous facts cited in Chapter II permit making the following

conclusion. If during direct stimulation of the cortex we record

from a given point of the cortex, at first during certain conditions

of stimulation, simple effects in the form of negative fluctuations

of potential, and then (in virtue of the fact that the stimulation

was intensified or it was quickened, or because of the fact that

the narcosis had abated, or because of local poisoning of the cortex

in the region of discharge or stimulation) the effects changed their

character, i.e. their sign was changed or additional fluctuations

appeared, then this change of character of effects needs to be treated

as an expression of the fact that certain new elements of the cortex

have been drawn into the reaction. In other words, as a result of

stimulation of the fibers of layer I of the cortex now not only the

dendritic plexus of the surface layers mainly is excited but excitation

occurs of certain complexes of neurons which were not excited in the

beginning (i.e. during other conditions of the experiment).

During deep narcosis the effects of the tetanic stimulation of

the surface of the cortex at a rhythm of 25-100 per second quickly

dwindle to nothing and the several-second duration of tetanization

does not lead to the arising of any electrical phenomena in the cortex

which might be observed by using a booster of alternating current.
Moreover, for a certain distance around the stimulating electrodes
depression is observed of the "spontaneous" electrical activity
which has been spoken of in detail in the preceding chapter (see
Figs. 19-21).

In easily narcotized animals the phenomena at first develop
the same as during deep narcosis: in the very first moments of
tetanization the effects attenuate sharply and in the course of
several seconds stimulation remains, as it were, ineffective.
However, after 5-15 seconds of stimulation a most intense rhythmical
electrical activity arises.

(Legend to Fig. 42, textpage 104: Rhythmical electrical
activity arising in a limited part of the cortex at tetanization
of the cortical surface. Cat No. 26, June 3, 1950. 2 hours after
the experiments the recordings of which are presented in Fig. 19,
A and B; surface nembutal sleep. The stimulating pair (P) and
the discharge electrode (E_1) are placed on the surface of the gyrus
suprasylvius; distance between them 6 mm. Intensity of stimulation
30 v. A - at first the effect of one shock of stimulation, then
at the moment indicated by the arrow stimulation is applied at a
frequency of 40 per second. B - 8 seconds from the start of stimulation.
C - 9 seconds from the start. D - 10 seconds from the start. E - end

of stimulation. After this at a distance of 10 mm. from the stimulating
electrodes the second discharge electrode E_2 was set up. F - scheme of
placement of electrodes. G - biopotentials are discharged simultaneously
from point E_1 (upper curve) and from point E_2 (lower curve); 15 seconds
from the start of tetanic stimulation at 40 per second, 30 v. (Roitbak,
1953a).)

Fig. 42 illus_trates this phenomenon. In this experiment the
stimulating electrodes and discharge electrode were placed on the gyrus
suprasylvius. The separate shocks of stimulation provoked a negative
slow potential with an amplitude of approximately 0.3 milliv. At the
moment indicated by the arrow, 60 milliseconds after application of a
single shock of stimulation, the frequency of stimulation was exchanged
for 40 per second (osc. A). After the initial effect, the negative
potential, each shock of stimulation began to produce a weak (50-60
micorov.) positive fluctuation; after several seconds of stimulation
these positive fluctuations almost dwindled to nothing. Then during
prolonged tetanization there suddenly arose slow negative fluctuations
of relatively great amplitude (0.4 milliv.) rhythmically following one
another, which proceeded not according to the rhythm of stimulation but
according to a far more infrequent rhythm: at first 14 per second,
then 12 per second, and after several more seconds of stimulation 10
per second (osc. B-D). At cessation of stimulation the rhythmical
electrical activity was broken, i.e. the arising of it was causally
connected with cerebral stimulation. The phenomenon described was

reproduced further on the preparation in question scores of times.

Thus, it is possible to say of the two phases of tetanization of the cortex: in the first quick abatement of effects and depression of the so-called "spontaneous" electrical activity are observed, in the second a most energetic rhythmical electrical activity arises.

Below are indicated the conditions for the arising of a given rhythmical electrical activity and certain of its characteristics.

1. The animal should be either without narcosis or under very light narcosis (active reflexes to tactile stimulations, liberating movements).

2. The stimulating shock should be of a certain minimal intensity; during a lesser intensity of stimulation the rhythmical electrical activity does not arise although activation of the surface layers of the cortex can occur.

3. The frequency of stimulation should be no lower than a certain limit: on lightly narcotized animals no lower than 25 per second; but on non-narcotized animals rhythmical electrical activity can arise even at a frequency of stimulation of 3 per second.

4. The greater the intensity of stimulation and its frequency, the shorter the latent period of the arising of rhythmical electrical activity, which can become equal to 1 sec. in all.

5. During uninterrupted stimulation the rhythmical activity gradually abates and ceases at approximately 60 seconds after the

moment of its arising.

6. It is possible to provoke rhythmical electrical activity
in any part of the cortex; on the other hand, it can be provoked at
a given point of the cortex during stimulation of various points of
the cortex and not of any one certain point.

7. For repeated obtaining of the phenomenon it is necessary to
observe intervals of 3 min. or more between the tetanic stimulations.

8. At interruption of stimulation that has provoked rhythmical
electrical activity an aftereffect is often observed (Fig. 44); the
electrical effects in the aftereffect are of an entirely different
character. During repeated tetanizations of the cortex with intervals
of 3-4 minutes the aftereffects become even more and more prolonged.

9. Rhythmical electrical activity, if it arises in the motor
area of the cortex, can be attended by movements arising long before
they set in. The arising of the focus of rhythmical activity in
other regions of the cortex is usually not attended by any motor or
vegetative reactions.

10. A phenomenon of a similar sort is not observed in
experiments on spinal cord either during protracted direct electrical
stimulations of the gray matter or during protracted stimulations
of the nerves (Beritov and Roitbak, 1950).

After the experiment considered (Fig. 42, osc. A-E) on gyrus

suprasylvius the second discharge electrode E_2 was placed 10 mm.

distant from the stimulating electrodes; the discharge electrode

E_1 was left at the first place (6 mm. distant from the stimulating

electrodes). During tetanic stimulation (40 per second) at first

both from E_1 and E_2 bioelectrical potentials were discharged (the

effects from E_2 having been altered somewhat by polarization).

After several seconds of tetanization the electrical effects both

at E_1 and E_2 dwindled almost to nothing. Then during prolonged

stimulation from E_1 slow negative fluctuations began to be discharged,

rhythmically following one another, and the phenomenon already

familiar to us arose; from E_2 at this time no additional effects

were discharged (osc. G). Thus, the rhythmical electrical activity

being considered arises in a limited focus of the cortex and cannot

spread through the cortex even within the limits of one convolution.

Usually rhythmical electrical activity is not registered at a distance

greater than 10 mm. from the stimulating electrodes (on narcotized

animals).

Rhythmical electrical activity arising during prolonged

tetanization of the cortex can bear a most diverse character, but

for a given preparation with fixed position of the stimulating and discharging

electrodes its character remains constant and tens of time in succession

it is possible to observe one and the same sterotypic reaction.

(Legend to Fig. 43, textpage 107: Rhythmical electrical
activity arising at tetanization of the cortical surface. Cat No.
55, July 7, 1953. The stimulating electrodes and the discharge
electrode are placed on the surface of the gyrus suprasylvius;
distance between them 6 mm. Intensity of stimulation 30 v. A -
in the beginning frequency of stimulation about 5 per second; at
the end of the oscillogram frequency of stimulation about 100 per
second. Each recording after recording A is an immediate continuation
of the preceding. E - end of stimulation at 100 per second and
change-over to a frequency of stimulation of 5 per second. The
long fluctuation s at a rhythm of about 30 per minute are evidently
governed by a respiratory pulsation of the brain.)

In Fig. 43, where each recording is an immediate continuation
of the preceding, the course of the development of rhythmical
electrical activity is seen. The separate shocks of stimulation
provoked a negative potential lasting about 10 milliseconds (i.e.
an elementary dendritic potential arose), after which a long positive
potential followed (osc. A). At switching to a stimulation frequency
of 100 per second only the first 4-5 shocks provoked an effect
(negative potential); in the course of the following three seconds
the stimulation remained apparently entirely inactive: only artefacts
of stimulation were recorded (osc. B). Next rhythmical electrical
activity arose in the form of negative fluctuations of potential.
The amplitude of the fluctuations in the course of the next 6 seconds

of tetanization constantly increased; the frequency of the rhythm
gradually diminished in the course of stimulation from 30 to 20 per
second (osc. C-E).

The phenomenon considered arises also during stimulation of
the white matter of the cortex, i.e. during the action of the afferent
impulses on the cortex (Fig. 46). Nevertheless, even during
tetanization of the cortical surface activation of its neuronic
elements proceeds by the "physiological route" and in our experiment
conditions by impulses of excitation of the fibers of the first layer
of the cortex directly stimulated.

A theoretical analysis permits concluding that the rhythmical
activity is linked with the activity of other neuronic elements than
those which are implicated during infrequent stimulations or at the
beginning of tetanization. During infrequent and weak stimulations
excitation in the system of fibers of layer I, evidently also at
participation of the neurons of layers I and II, extensively over-
flows through the cerebral cortex, everywhere stipulating the
arising of regional processes of excitation in the neuronic elements
of the surface layers of the cortex. This finds its electrical
expression in the fact that during infrequent stimulations of any
point of the cortex from any other point of the surface of the
hemisphere negative fluctuations of potential are usually discharged.

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Rhythmical electrical activity is stipulated certainly by
excitation of certain complexes of intracortical neurons, evidently
not efferent but neurons with short axons which are just as numerous
in the cortex. They must be those complexes of neurons on which
impulses from the nerve elements first excited (by direct stimulation
of the cortical surface) act to a subthreshold extent. During
prolonged tetanization heightening of their excitability occurs and,
when it reaches a certain critical magnitude, these neurons act that
possess, as seen, an extraordinary capacity for summation. Besides
an extraordinary capacity for summation, these cortical neurons, as
it is possible to conclude on the basis of the data already stated,
are very susceptible to the effect of narcotics and are characterized
by quick fatiguability.

By using Nissl and Cajal's method for staining, we succeeded
in catching the fine morphological changes of the neuronic elements
of the cortex in the region of the focus of rhythmical electrical
activity that is created by electrical stimulation of the cortical
surface (Cholokashvili and Roitbak, 1955). Changes are observed in
the fibers of layer I (which are restained and more intensively convoluted
than in the normal), in the top dendrites of the pyramids (which are
rough, i.e. the contour of which is less smooth than ordinary) and
in the bodies of the neurons, mainly of cortical layers II and III

(between the nucleus and the protoplasm of the cell a clear boundary is worn, the Nissl bodies protrude worse, more often tiny vacuoles are encountered). These structural changes, not residual imprints of the pathological, are evidently connected with the process of excitation of the corresponding elements. Thus, the histological data obtained fully agree with the ideas developed on the mechanism of activation of the cortex during stimulation of its surface.

(Legend to Fig. 44, textpage 109: Electrical activity of the cortex at the time of its tetanic stimulation and in the aftereffect. Cat No. 38, Jan. 9, 1951. 2 pairs of stimulating (P_1 and P_2) and 2 discharge electrodes (E_1 and E_2) are placed on the surface of the gyrus suprasylvius. The biopotentials are discharged from point E_1 (upper curves) and from point E_2 (lower curves). A - electrical activity 6 seconds after beginning of stimulation through electrode P_2 with a frequency of 50 per second, 25 v. (P^2_{50}). B - aftereffect; section 1.5 seconds after cessation of P^2_{50} stimulation; C - after 3 seconds, D - after 11 seconds; E - scheme of location of electrodes with indication of distance between them in mm. in experiments A-D. F - scheme of location of electrodes in experiments G and H. G - electrical activity 4 seconds after the start of the combination of P_1 stimulations at the rate of 50 per second (P^1_{50}, 25 v.) and P_2 at a rhythm of 50 per second (P^2_{50}, 25 v.). H - the aftereffect subsequent to cessation of P^1_{50} + P^2_{50} stimulation (Roitbak, 1953a).)

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Why is the rhythmical electrical activity limited to certain
foci? Why does the excitation spread not occur over the whole cerebral
cortex? Evidently another process, limiting it, exists side by side
with the excitation. Experiments, the recordings of which are presented
in Fig. 44, give basis for such a conclusion. At time of tetanization
of the cortex the rhythmical electrical activity that has arisen in
a certain part of the cortex does not spread to a region several
millimeters distant; however, at cessation of the tetanization in the
aftereffect the activity spreads to this other region also. Thus, at
time of tetanic stimulation that has provoked and maintained a focus
of rhythmical activity a process occurred that limited the spread of
this activity. Apparently this is a braking process. Ukhtomskii
(1926) had already come to the conclusion that "during electrical
stimulation of the cortex it is fitting to think of an active localization
of excitation... in counterbalance to the action of the branching currents.
However, this active localization can be accomplished only by a braking
action." By the way, electrical effects during local strychnine poisoning
of the cortex is limited strictly by the place of poisoning, and the
process of inhibition was the reason indicated for this phenomenon
(Beritov, 1948). Below facts are presented that indicate the inhibition
not only limits the focus of rhythmical activity but also gives it no
opportunity to develop to its full strength and weakens this activity

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in the very focus of its arising.

The following experiments were set up with a combination of stimulations of two points of the cortex and with registration of the electrical potentials arising.

The results of one such experiment are presented in Fig. 45 (textpage 110: Bioelectrical phenomena at the focus of heightened excitability created in the cerebral cortex. Cat No. 24, May 20, 1950. The first pair of stimulating electrodes (P_1) is established at the posterior pole of the gyrus suprasylvius, the second (P_2) at the anterior pole; the discharge electrode (E) is located in the middle of the convolution. A - stimulation through electrodes P_1 at a rhythm of 9 per sec. (P^1_9) and addition of stimulation through electrodes P_2 at a rhythm of 50 per sec. (P^2_{50}); intensity of stimulation 30 v. B - immediate continuation of A. C - continuation of a combination of stimulations $P^1_9 + P^2_{50}$ 12 seconds after the moment of addition of P^2_{50}. D - cessation of P^2_{50} stimulation during uninterrupted stimulation by P^1_9. E - 3 seconds after cessation of combination of stimulations. F - after a further 15 seconds. G - after a further 10 seconds; cessation of P^1_9 stimulation. H - scheme of location of electrodes (Roitbak, 1953a).).

The following was the set-up of this experiment. The first pair of stimulating electrodes (P_1) was located at the rear pole of the gyrus

suprasylvius, the second pair of stimulating electrodes (P_2) was

established on the anterior pole of this convolution, around the

sensory-motor region. The discharge electrode (E) was located midway

between the two stimulating pairs.

Stimulation through P_1 at a rhythm of 9 per sec. (P^1_9) provoked

at point E simple effect: in response to each shock of stimulation

a negative fluctuation arose of small amplitude, on the descending

limb of which there was a weakly expressed additional fluctuation

(Fig. 45, osc. A). Thus the P_1 stimulation by itself caused activation

of the dendritic plexus of the upper layers mainly under the effect

of impulses from the stimulated fibers of layer I. During prolonged

stimulation of the P_1 point at a rhythm of 9 per second it was

possible to observe progressive reduction of the amplitude of the

biopotentials, their character not having altered.

After 20 shocks of P^1_9 stimulation a tetanic stimulation was

added through the P_2 electrodes at a rhythm of 50 per second (P^2_{50}).

In the first moments of tetanization each shock of P^2_{50} caused a

negative fluctuation. On the background of this effect only weak

P^1_9 stimulation was found, reflection was hardly to be observed (osc.

A). Such a picture was observed in the course of several seconds of

tetanization, at which time progressive abatement of the P^2_{50} effects

occurred (osc. B).

Thus, in the first seconds of tetanization around the point
of the cortex being stimulated inhibition develops which is expressed
not only in depression of the "spontaneous" electrical activity,
but also in attenuation or falling out of the effects of stimulation
of other regions of the cortex. Several seconds after P^2_{50} tetanization,
when the shocks of this stimulation readjusted to provoke their own
direct effects, energetic rhythmical activity arose (osc. C). However,
the effect that arose differs from those which we have considered up
to now: slow negative potentials arise at P^1_9 rhythm, at the rhythm
of combined stimulation, (and) in response to the shocks of this
stimulation.

The effects which arose in response to P^1_9 stimulation at the
time of P^2_{50} tetanization differed characteristically from the usual
effects of P^1_9 - at each shock of stimulation with a greater latent
period (about 20 seconds) a slow negative potential of larger amplitude
set in; direct effects of P^1_9 stimulation, which were provoked by this
stimulation prior to the combination of stimulations, arose now too,
but their amplitude was reduced. In other words, the effects stipulated
by the activity of the cortical neurons were extraordinarily intensified.
Thus, the significance of such phenomena: as a result of tetanic
stimulation (P^2_{50}) the excitability is extremely heightened in a certain
group of cortical neurons; at this time the stimulation of another point

of the cortex (P^1_9) provokes their excitation.

At cessation of (P^2_{50}) tetanization of the P^1_9 effects begin
to intensify (osc. D) and their growth occurs in the course of 0.5
seconds: evidently at the time of P^2_{50} tetanization the process
of inhibition took place and this inhibition had a prolonged
aftereffect. At P^2_{50} cessation the P^1_9 effects changed in character
too: they became more prolonged, double and triple effects appeared,
and after the negative complex a considerable positive deviation
arose (osc. E-G). Infrequent stimulations of the P_1 point for a
period of tens of seconds at cessation of the combination with the
tetanic stimulation of the P_2 point continued provoking the complex
effects described. Next, these effects simplified, abated, and
ceased to be provoked. The P^1_9 again provoked its direct effects
in the form of simple negative potentials that set in with a
negligible latent period but considerably lesser amplitude than at
the beginning of the experiment prior to annexation of P^2_{50}; a
rest of 30-60 seconds was sufficient for these effects to be restored
to their initial magnitude.

Thus, elevated excitability of a certain complex of neurons
is prolonged a long time after cessation of tetanic stimulation of
the cortex and can be demonstrated at stimulation of another point
of the cortex located at a considerable distance from the point

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subjected to tetanization. (Footnote: Rosenblueth and Cannon (1942)

cite in their work an experiment (on monkey), the results of which

amount to the following. Separate shocks of stimulation applied to

the motor area of the cortex for a back paw (foot) provoked certain

electrical effects in the motor area for a front paw (hand). After

repeated tetanic stimulations of the motor area of the facial muscles

convulsive electrical activity arose that spread over a vast region

of the cortex, including the motor area for a front paw. Separate shocks

of stimulation provoked at the time of convulsive electrical activity

and within a certain time after its cessation intensified electrical

effects similar to the components of the convulsive activity. However,

Rosenblueth and Cannon, not having attached any special importance to

these facts, did not subject them to analysis.)

In Fig. 45 it is seen that infrequent stimulations (P^1_9) in

consequence of union with tetanic stimulation (P^2_{50}) begin to cause

new complex effects which, as compared with the initial effects of

P^1_9 stimulation, are completely altered, such as would occur if part

of the cortex was poisoned by strychnine (see Fig. 45, E and Fig. 23, B).

On the other hand, infrequent shocks of stimulation of another, often

remote, part of the cortex, producing new effects and exciting neurons

found in a state of heightened excitability, produce direct effects of

their own too. For instance, in Fig. 45, A, it is seen that prior to

the combining of stimulations P^1_9 and P^2_{50} in response to each shock of stimulation a negative potential was registered, which arose after an artefact of stimulation, i.e. impulses of excitation proceeding from point P_1 activated at the point of discharge mainly the synaptic endings to the top dendrites, but they also reached the intracortical neurons apparently of layer II (see textp. 65) sometimes causing excitation of a certain small number of them, which was expressed in the appearance of a small "hump" on the descending limb of the dendritic potential. When the focus of rhythmical electrical activity was linked with the tetanization of point P_2 in the discharge part of the cortex, then the shocks of stimulation of P^1_9 began to produce larger slow potentials that set in with a latent period of about 20 milliseconds; but in the latent period of these effects after an artefact of stimulation the previous simple negative potentials arose (Fig. 45, C-G); their amplitude was apparently reduced in consequence of the prolonged P^2_{50} tetanization (compare Fig. 30, A and B).

Hence, it is possible to make the following conclusion. In the presence of a focus of heightened excitability impulses of excitation from extraneous sources are not deflected at this focus; impulses of excitation during secondary stimulations proceed there and by the same pathways as when it is not a focus of heightened

excitability. They thereby produce an intense bioelectrical reaction
of elements which are found in a state of heightened excitability (to
which these impulses proceed even prior to the creation of a focus
of heightened excitability, but they could at that time show only a
subthreshold effect on them), however, as before, they produce also
that initial bioelectrical reaction which is characteristic to them
in normal conditions.

(Legend to Fig. 46, textpage 113: Bioelectrical phenomena at
source of heightened excitability created in the cerebral cortex.
Cat No. 33, July 16, 1950. First pair of stimulating electrodes (P_1)
placed on surface of gyrus suprasylvius, second pair $(P_2$, microelectrodes)
thrust into the white matter under the cortex at a distance of 10 mm.
from P_1; discharge electrode (E) placed on the surface of the brain
between P_1 and P_2. A - stimulation through P_1 electrodes at a rhythm
of 10 per second (P^1_{10}) and addition of stimulation through P_2
electrodes at a rhythm of 11 (P^2_{11}); the shocks of stimulation of P_2
are indicated by arrows. B - continuation of the combination of
stimulations and cessation of P^2_{11} stimulation. C - P^1_{10} stimulation
and addition of P^2_{50} stimulation. D - continuation of combination of
$P^1_{10} + P^2_{50}$ stimulations 15 seconds after the moment of the annexation
of P^2_{50}. E - cessation of P^2_{50} stimulation (moment of cessation
indicated by arrow) during uninterrupted P^1_{10} stimulation. F,G -
continuation of P^1_{10} stimulation. H - scheme of arrangement of
electrodes (Roitbak, 1953a).)

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In Fig. 46 the results are presented of a somewhat modified experiment. The first pair of stimulating electrodes (P_1) was placed on the surface of the brain. At a distance of 4 mm. from it on the surface of the brain the discharge electrode (E) was located. Two isolated microelectrodes served as second pair of stimulating electrodes. They were sunk deep into the white matter under the cortex at a distance of 6 mm. from the discharge electrode. Thus, the discharge electrode was between the electrodes that stimulated the surface of the brain and the electrodes that stimulated the white matter (see Fig. 46, H). Stimulation through P_1 with a frequency of 10 per second (P^1_{10}) produced negative fluctuations of the biopotential with an amplitude of about 0.75 milliv. (osc. C, the beginning). At addition to P^1_{10} of a frequent tetanic stimulation through P_2 at 50 per second (P^2_{50}) complete inhibition occurred of the effects of P^1_{10} stimulation (osc. C). After 7 seconds of a combination of the stimulations an energetic rhythmical electrical activity arose at the rhythm of P_1 (osc. D). Thus, this stimulation "tied" its own rhythm onto the neuronic elements of the focus of heightened excitability. In other words, P^1_{10} on a background of the tetanic P^2_{50} stimulation began to yield complex effects of a character completely different from those which were produced by it prior to union of stimulations, namely the P^1_{10} began to produce

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positive fluctuations of potential, solitary and double. Thus, prior
to the combination of stimulations $(P^1_{10} + P^2_{50})$ the P^1_{10} stimulation
was not markedly reflected on certain nerve elements located, as we
have a right to think, in the deep layers of the cortex. As a result
of the union with the tetanic stimulation of P^2_{50}, the P^1_{10} stimulation
becomes capable of exciting them not only at the time of P^2_{50} tetanization,
but also at a time long after its cessation (osc. E - G).

On the basis of the experiment considered with stimulation of
the white matter it is possible to conclude that the focus of rhythmical
electrical activity can be created by the action of afferent impulses
that come to the cortex.

If the P^1_{10} stimulation (stimulation of the surface of the brain)
stimulation of the white matter was added at a rhythm of about 10 per
second $(P^2_9$ or $P^2_{11})$, then depression of the P^1_{10} effects was observed:
the negative fluctuations produced by stimulation of the surface of
the brain abated, their degree of abatement having been determined by
the time interval between shocks P_1 and P_2 (Fig. 46, osc. A and B);
at cessation of P_2 stimulation the P_1 effects gradually increased
and after a certain time reached their initial magnitude.

Thus, we encounter extraordinary alterability and numerous
phenomena. In the first place, stimulation of a given point of the
cortex can produce different effects depending on the phase of its
action (2 phases of tetanization of the cortex). In the second
place, the stimulation of a given point of the cortex stipulates

different effects depending on the frequency of stimulation.

For instance, in the case just considered the stimulation through the

P_2 electrodes at a rhythm of 11 per second produced only depression

of the effects of P_1 stimulation; stimulation through P_2 at a rhythm

of 50 per second in the second phase of tetanization stipulated

complete inversion of the effects of P_1 stimulation. (This important

question will be examined again in a special way.)

(Legend to Fig. 47, textpage 115: Bioelectrical phenomena at

focus of heightened excitability created in the cerebral cortex. Cat

No. 34, Oct. 24, 1950. Stimulating electrodes and discharge electrode

are located on the surface of the gyrus suprasylvius. The first

discharge electrode (E_1) is found at a distance of 4 mm, the second

(E_2) at a distance of 10 mm. from the stimulating electrodes. Bio-

potentials are discharged simultaneously from the E_1 point (upper

curves) and from the E_2 point (lower curves). A - beginning of

stimulation at a rhythm of 12 per second; intensity of stimulation

30 v. B - continuation of stimulation at a rhythm of 12 per second

and shift-over to a stimulation frequency of 80 per second. C - after

3 sec., D - after 8 sec., E - after 10 sec., F - after 13 sec. of

uninterrupted tetanic stimulation. G - scheme of arrangement of

stimulating and discharge electrodes (Roitbak, 1953a).)

In the experiments the recordings of which are presented in

Fig. 47 the stimulating electrodes and 2 discharge electrodes were

placed on the gyrus suprasylvius; the first discharge electrode (E_1)

at a distance of 4 mm., the second (E_2) at a distance of 10 mm. from

the stimulating electrodes (see Fig. 47, G). At first stimulation

was applied at a frequency of 12 per second (osc. A). At point E_1

in response to each shock of stimulation double effects arose: after

the first negative fluctuation (1 milliv.) that lasted about 10

milliseconds, even prior to its completion, a second set in (0.8

milliv.), about 40 milliseconds in length. At point E_2 double negative

fluctuations (0.19 and 0.3 milliv.) also arose; in the course of

stimulation these effects intensified somewhat. At shifting the

frequency of stimulation to 80 per second (osc. B) the effects in

E_1 and E_2 as early as after the first shocks of tetanic stimulation

changed their sign and then quickly attenuated. After 3 seconds of

tetanization additional fluctuations began to arise, owing to which

the curves acquired a wavy character (osc. C). After 8 seconds of

tetanization at both points of the cortex a rhythmical electrical

activity arose: from E_1 positive fluctuations of considerable

amplitude (up to 0.6 milliv.) were discharged at a rhythm of about

25 per second; from E_2 negative fluctuations (up to 0.35 milliv.)

were discharged at the same rhythm (osc. D). On the 10th second of

tetanization the rhythmical activity lost its regular character

(osc. E). On the 13th second at point E_1 after regular intervals there

began to arise groups of 3-4 positive fluctuations; at point E_2

in respect to each such group a single negative fluctuation of

potential of great length (osc. f) arose. These electrical effects

set in at 2-3 times a second, and such activity was observed for

a period of a further 15 seconds of tetanization.

Thus, the rhythmical electrical activity that arose as a

result of tetanic stimulation of the cortex can be changed considerably

in its character in the course of prolonged tetanization: each given

moment of tetanization is distinguished from the preceding or the

subsequent one in the sense of the composition of the complex of

neurons being activated.

It has already been said that stimulation of a given point

of the cortex can produce effects different in character depending

on the frequency of stimulation. The experiment just considered is

a good illustration of this aspect. During stimulation of a given

point of the cortex at a rhythm of 12 per second at point E_1 preeminent

activation of elements of the surface layers of the cortex occurs;

certain complexes of neurons of the deep layers are thereby excited,

however long this stimulation lasts. However, stimulation (of the

same intensity) of the very same point of the cortex draws these new

neuronic complexes into reaction during an increase of its frequency

to 40-80 per second.

Furthermore, during tetanic stimulation of the cortex rhythmical

activity arises within the limits of a relatively large territory.
Moreover, at different points of this region rhythmical activity can
have a different character, which assumes the participation in it of
neuronic elements of a different sort of origin. In the given case
(Fig. 47) local tetanic stimulation of the surface of the brain led
to the arising of rhythmical effects even at point E_1, which is
found at a distance of 4 mm. from the stimulating electrodes, and
at point E_2, at 10 mm. from the part stimulated. However, at E_1
positive, and at E_2 negative fluctuations of the electrical potential
arose. In other words, at the center of the focus of rhythmical
activity excitation mainly of the neurons of the deep layers of the
cortex occurred; at the periphery excitation of elements of the
surface layers predominated (i.e. mainly dendritic offshoots in
layers I and II). (Footnote: If relationships of such sort prove
regular, then, inasmuch as activation of the surface layers is
connected with depression of the "spontaneous" electrical activity,
it would be very tempting to admit that activation of the surface
layers of the cortex along the periphery of the focus of this activity
is the reason for restriction of the rhythmical activity. At that
time also it would be possible to cite these data as an electrophysiological
demonstration of the following position of Pavlov: "The point of
concentration of stimulation to a more or less extent is surrounded

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by a process of inhibition" (Pavlov, 1932).)

At examination of Fig. 47 we dwelled on the fact that as a result of several-second tetanization (80 per second) of the cortex at points E_1 and E_2 complex rhythmical electrical activity arose. At the 30th second of tetanization the frequency of stimulation was shifted to 12 per second (Fig. 48, osc. A). (Legend to Fig. 48, textpage 117: Continuation of the experiments the recordings of which were presented in the preceding illustration. A - after 30 seconds of uninterrupted stimulation at a rhythm of 80 per second, the frequency of stimulation is changed momentarily to 12 per second. B - direct continuation of recording A. C - after 3 seconds, D - after 6 seconds, E - after 9 seconds, F - after 15 seconds of uninterrupted stimulation at a rhythm of 12 per second. After this the frequency of stimulation was again shifted to 80 per second. G - after 13 seconds of tetanization the frequency of stimulation of 80 per second shifts to 12 per second.) Right at the same rhythm of stimulation there began to arise at point E_1 positive fluctuations (50 milliseconds) and at point E_2 negative (80 milliseconds) of electrical potential, the amplitude of which equaled +0.9 and -0.65 milliv. Thus, after tetanization infrequent stimulations of the same point of the cortex produced at E_1 entirely distorted effects; as regards the effects of point E_2, then an extraordinary intensification

occurred of a second negative fluctuation (compare Fig. 47, A and

Fig. 48, A).

As seen from examination of Figs. 47 and 48, infrequent stimula-

tions of a given point of the cortex that impart change to the tetanic

stimulation and have created a focus of rhythmical electrical activity

produce in this focus effects the same in character as there were at

the time of tetanization, i.e. infrequent shocks of stimulation excite

the same complexes of neurons as stipulate by their activity the

arising of rhythmical electrical activity, complexes which were not

excited by infrequent shocks of stimulation prior to tetanization.

Thus, elevated excitability of the neurons of our focus can be

demonstrated both during stimulation of other points of the cortex

(Fig. 45) and during stimulation of the same point of the cortex,

the stimulation of which is stipulated by the arising of this focus.

Finally, these phenomena are of one order.

Attention is again drawn to Fig. 48 because at cessation of

tetanization the effects of infrequent stimulations increased during

the first second, reaching amplitudes of -0.9 and +1 milliv. (osc. B).

Progressive abatement of them began after 5 seconds, the attenuation

of the positive fluctuations (effects at E_1) having proceeded more

quickly than the negative (the effects at E_2), and after 9 seconds

from the moment of cessation of stimulation from E_1 negative fluctuations

were again discharged, i.e. the effects acquired the usual character,

and from E_2 there were again discharged negative fluctuations considerably

intensified as compared to the normal (osc. E). Shortly after this the

frequency of stimulation again was changed over to 80 per second; as

a result of several-second tetanization, weak rhythmical activity arose

(osc. G); infrequent stimulations, imparting frequent change, produced

extremely attenuated effects ordinary in character (compare Fig. 48, G,

and 47, A). Usually, as already said, repeated tetanic stimulations

of the cortex do not cause rhythmical electrical activity if the interval

between the stimulations is less than 3-4 minutes. Thus, in order to

obtain the phenomena considered by us, rhythmical electrical activity

of certain elements at time of tetanization of the cortex and prolonged

elevation of their excitability at cessation of tetanization, the part

of the cortex in question should be exhausted by previous activity.

Experiments similar in nature are presented in the recordings

of Fig. 49 (textpage 119: Bioelectrical phenomena at a focus of

heightened excitability created in the cerebral cortex. Cat No. 30,

July 1, 1950. The stimulating electrodes are thrust in under the

cortex in the region of the posterior pole of the gyrus suprasylvius;

the discharge electrode is placed on the surface of this convolution

at a distance of 10 mm. from the stimulating electrodes. A - effect

of stimulation at a rhythm of 10 per second (30 v.). B - 0.8 sec.

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from the start of stimulation at a rhythm of 50 per second. C -

25 seconds from the start of stimulation at a rhythm of 50 per

second and shift to a frequency of stimulation of 10 per sec. D -

0.3 sec. after C. E is 1 minute after D. F - effect of stimulation

at a rhythm of 10 per sec. before poisoning. G - effect of the

same stimulation 17 minutes after local strychnine poisoning (1%

solution) of the cortex under the discharge electrode.). In these

experiments the stimulating electrodes (microelectrodes) were

driven into the gyrus suprasylvius to the white matter; the discharge

electrode was established on the surface of the cortex at a distance

of 8 mm. from the stimulating pair. Shocks of stimulation at

infrequent rhythm caused at the discharge point two-phase fluctuations

of small amplitude: at first a positive, then a negative fluctuation

arose (Fig. 49, osc. A). Evidently activation of the cortex occurred

by means of fibers that entered from the white matter: as known,

afferent and callosal fibers terminate mainly in cortical layers III

and IV. At arrival in the cortex of a volley of excitation impulses

along these fibers first of all the elements of these layers are

excited, which is expressed in the fact that at discharge from the

surface of the corresponding part of the cortex a positive fluctuation

of potential is registered, after which, when the functional state

of the cortex is good, a negative fluctuation follows.

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With a shift-over to frequent tetanic stimulation (osc. B), at the rhythm of stimulation small positive fluctuations began to arise. After several seconds of tetanization a complication of effect set in: at irregular rhythm slow fluctuations of greater amplitude began to arise (osc. C). With transition after this to infrequent stimulations, it was observed that the latter began to cause intense complex bioelectrical effects (osc. D). These effects are not intensified initial effects (i.e. effects which were caused before tetanization of the white matter). On the contrary, the initial complex (+-) abates, but after it with a considerable latent period first a positive, then a most intense negative potential arise. After many seconds of stimulation these new additional potentials ceased to arise, and each shock of stimulation caused an effect the same in character as at the very beginning of the experiment, only highly attenuated.

Intense additional potentials, which arose after tetanization, set in with a greater latent period (of the order of 20 milliseconds), which indicates participation of cortical neurons intermediate in their origin.

If in the preceding case (Fig. 48) the activity of the new elements as a whole masked the initial bioelectrical effect, since the new elements were excited directly in response to stimulation, then

in the case in question the new elements were drawn into the reaction
after the end of the initial bioelectrical reaction that set in
immediately in response to stimulation.

In the recordings just cited it is likewise seen that the so-
called "spontaneous" slow potentials that arise during tetanization
are considerably smaller in amplitude than the slow additional
potentials arising in response to shocks of infrequent stimulation,
incessantly changing; they are not only smaller, but they are also
variable in amplitude and length.

As known, strychnine has a characteristic capacity for
increasing the excitability of neuronic elements. Consequently,
it would be possible to expect that during local strychnine poisoning
of the cortex phenomena would arise like those considered above,
which were treated as a consequence of heightened excitability of
cortical neurons.

Some time after the experiments described with tetanization
of the cortex (Fig. 49, A-D) the area under the discharge electrode
was poisoned with 1% strychnine solution. Immediately before
poisoning, stimulation of the white matter at a rhythm of 10 per
second produced the effects already described above, consisting of
a brief positive fluctuation, after which a negative followed of
small amplitude but greater length, sometimes complicated by additional
"humps" (osc. F). After poisoning, the same stimulation began to
produce new effects: after the initial first complex, attenuated

in amplitude, a positive fluctuation arose with a latent period of
20-25 milliseconds, after which a most energetic negative potential
followed (osc. G). Thus, as a result of strychninization of the
discharge point of the cortex during stimulation effects arose like
those which began to arise in response to this stimulation after
tetanization of several seconds (compare osc. D and G). A similarity
is discovered also in such a detail as that in both cases additional
energetic potentials arise in response not to each shock of stimulation
but to each second or third shock.

Since it is well known that a heightening of the excitability
of the intermediate neurons lies at the base of the change of
bioelectrical effects under the action of the strychnine, then the
facts cited can serve as demonstration of the fact that the phenomena
being considered, connected with prolonged tetanic stimulation of the
cortex, govern heightening of the excitability in the new additional
complexes of the intracortical neurons which usually are not excited
in response to infrequent shocks of stimulation.

On the basis of the facts just considered it is possible to
conclude that the arising of complex effects new in character after
tetanization is connected, at least in the case in question, with
change of the state of the neurons in the region from which those
effects were recorded and not in the region of the application of

stimulation, because the white matter was stimulated and, secondly,

an essentially similar result was obtained at local strychnine poisoning

in the discharge region. As already said, strychnine convulsive effects

are not limited strictly to the region of poisoning.

As already said, during combination of stimulation of two points

of the cortex (one point is stimulated tetanically, the other with

relatively infrequent shocks) rhythmical electrical activity arises,

and the rhythm frequency is determined by the frequency of the second

infrequent stimulation: this stimulation "imposes" its rhythm on the

neuronic elements of the focus of heightened excitability.

(Legend to Fig. 50, textpage 121: Bioelectrical phenomena at

the source of heightened excitability created in the cerebral cortex.

Cat No. 38, Jan. 9, 1951. Two pairs of stimulating (P_1 and P_2) and

2 discharge (E_1 and E_2) electrodes were placed on the surface of the

gyrus suprasylvius (see scheme). The biopotentials are discharged

from point E_1 (upper curves) and from point E_2 (lower curves). A -

stimulation through electrodes P_1 at a rhythm of 10 per second (P^1_{10})

and annexation of stimulation through electrodes P_2 at a rhythm of

50 per second (P^2_{50}). The intensity of stimulation of P_1 and P_2 is

25 v. B - length of combination of stimulation $P^1_{10} + P^2_{50}$ 9 seconds

after moment of annexation of P^2_{50}. C - stimulation through electrodes

P_2 at a rhythm of 50 per second (P^2_{50}). The 11th second after beginning

of tetanization. D - end of tetanization by P^2_{50} and beginning of

stimulation by P^1_{10}; moment of application of first shock through

electrodes P_1 is indicated by arrow. E - scheme of arrangement of

electrodes (Roitbak, 1953a).)

The set-up of the experiments, the recordings of which are

presented in Fig. 50, was more complex than those described up to

now: here registrations were made of the biopotentials of two

points of the cortex during combination of stimulations of two

points of the cortex (Fig. 50, E). Stimulation of the anterior

pole of the gyrus suprasylvius at a frequency of 10 per second

(P^1_{10}) causes at point E_1 (5.5 mm. from P_1) negative fluctuations

of potential and at point E_2 (19.5 mm. from P_1, the posterior pole

of the convolution) positive fluctuations of potential. At the

combining of the tetanic stimulation of the middle part of the gyrus

suprasylvius with a frequency of 50 per second (P^2_{50}) the P^1_{10}

stimulation becomes for a long time inactive, as it were (osc. A).

Several seconds after combining the stimulations rhythmical electrical

activity arises at point E_1 and E_2. From E_1 positive fluctuations

are registered and from E_2 negative fluctuations (osc. B). Thus,

we again are confronted with facts indicative that at the periphery

of a focus of intense rhythmical activity of the elements of the deep

layers of the cortex essential activation of the surface layers occurs.

The rhythm of the negative fluctuations at E_2 and of the positive at

E_1 is determined by P_1 stimulation: fluctuations arise in response
to shocks of this stimulation; from time to time double effects (osc. B)
arise to the shocks of P_1 stimulation.

P^2_{50} stimulation in itself causes rhythmical electrical activity
of a different character (osc. C): at uninterrupted stimulation groups
of 4-5 positive fluctuations arise periodically separated from one
another by 1/5 of a second pauses.

At cessation of the tetanic stimulation that produced rhythmical
electrical activity there often occurs a more or less prolonged after-
effect. The bioelectrical potentials at the time of the aftereffect
are usually similar in character to the bioelectrical potentials at
the time of tetanic stimulation (osc. D; see also Fig. 44). It is
possible to think that the aftereffect is stipulated by the activity
of the same neuronic elements as are excited at the time of tetanization.

If at cessation of tetanization of a given point of the cortex,
at the time of the aftereffect a relatively infrequent stimulation is
applied to another point of the cortex, then the aftereffect can be
broken and effects at the rhythm of the stimulation applied (osc. D)
begin to arise. (Footnote: Aftereffects at cessation of tetanization
lasted ten seconds and had the same character as in the recordings
of Fig. 44, which were made on the same preparation.) Thus, additional
stimulation can "control" the activity of the neuronic elements at the
focus of heightened excitability, determining the rhythm of their
excitation not only during its combination with tetanic stimulation

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that created this focus, but also at the time of the aftereffect,

when the rhythm of the arising bioelectrical potentials is determined

by influences not always yielding to calculation.

(Legend to Fig. 51, textpage 122: Bioelectrical phenomena at

focus of heightened excitability created in the cerebral cortex.

Non-narcotized rabbit. March 10, 1951. Breathes through cannula in

trachea. Through an opening in the bone and in the dura mater 2 pairs

of electrodes were placed on the surface of the cortex: in the motor

and optical regions. The biopotentials are discharged from the motor

region; stimulation is applied to the optical region. The intensity

of stimulation is 30 v. A - stimulation at a rhythm of 5 per second.

B - aftereffect at cessation of stimulation. C - repeated stimulation

at a rhythm of 5 per second at the time of the aftereffect. D -

immediate continuation of recording C. E - aftereffect after cessation

of stimulation.)

The phenomenon described is observed too in experiments on non-

narcotized animals. Fig. 51 serves as illustration for this position,

in which are presented recordings obtained in experiments on non-

narcotized tracheotomized rabbit. The discharge pair of electrodes

was set up on the motor area of the cortex, the stimulating electrode

on the optical area. Each shock of stimulation at a rhythm of 5 per

second produced in the motor area a definite electrical effect (osc. A);

at this time no motor or vegetative reactions arose. At cessation

of stimulation a prolonged aftereffect was observed in the form of

slow potentials at a regular rhythm of about 7 per second (osc. B).

Stimulation, when it was applied at the time of the aftereffect,

broke it off: effects began to arise at the rhythm of stimulation

(osc. C and D); at cessation of stimulation the aftereffect described

above again occurred (osc. E).

At combination of stimulations of two points of the cortex a

more intense rhythmical electrical activity usually arises than at

stimulation of one of these points. This also refers to cases when

tetanic stimulation is applied to the first point but the second

point is stimulated by comparatively infrequent electrical shocks

(Fig. 52, B and G). It is possible to think that during combination

of $P^1_{10} + P^2_{50}$ a greater number of the neuronic elements of a given

complex is implicated in reaction than in the case of isolated P^2_{50}

stimulation. Thus, additional P^1_{10} stimulation not only stipulates

the excitation rhythm of neurons of the dominant focus but also

evidently increases the number of focal elements excited.

If at combination of $P^1_{10} + P^2_{50}$ stimulations rhythmical electrical

activity arises, then at cessation of P^2_{50} stimulation the P^1_{10} stimulation

continues to excite usually for a period of tens of seconds the neuronic

elements of the focus of heightened excitability that has arisen. If

at combination of $P^1_{10} + P^2_{50}$ stimulations it does not lead to the

arising of rhythmical electrical activity, then at cessation of P^2_{50}

stimulation the P^1_{10} stimulation produces its own ordinary effects

as it did prior to union with the tetanic stimulation. If at

combination of $P^1_{10} + P^2_{50}$ a rhythmical electrical activity arises

and the combination of stimulation lasts tens of seconds before the

disappearance of rhythmical electrical activity, then at cessation

of P^2_{50} stimulation the P^1_{10} stimulation produces its own usual

effects, only greatly attenuated in comparison with those which it

provoked prior to union of the stimulations. Thus, infrequent P^1_{10}

stimulation at combination with the tetanic P^2_{50} stimulation does

not produce excitation of a given complex of neurons so long as

their excitability has not yet reached a certain critical magnitude

at which they are capable of being rhythmically excited under the

action of impulses of any origin that arrive in the cortex or if

this high excitability of theirs has already fallen because of

exhaustion.

(Legend to Fig. 52, textpage 124: Bioelectrical phenomena at

a focus of heightened excitability created in cerebral cortex. Cat

No. 37, Jan. 7, 1951. The first pair of stimulating electrodes (P_1)

was placed on the rear pole of the gyrus suprasylvius, the second

(P_2) on the anterior. The discharge electrode (E) was placed at the

middle of the convolution. A - stimulation through P_1 electrodes

at a rhythm of 10 per second (P^1_{10}) and combination of stimulation

through electrodes P_2 at a rhythm of 50 per second (P^2_{50}). The

intensity of stimulation was 25 v. B - continuation of combination

of stimulations 10 seconds after moment of annexation of P^2_{50}. C -

cessation of P^2_{50} stimulation during uninterrupted P^1_{10} stimulation.

D - continuation of recording C. E - effect of P_1 stimulation after

10 seconds' interruption in its action; stimulation was interrupted

several seconds after recording D. F - effect of P_1 stimulation

after 20 seconds of interruption in its activity. G - P^2_{50}

stimulation; 12th second from start of tetanization. H - end of

P^2_{50} tetanization and beginning of P^1_{10} stimulation. I - scheme of

arrangement of electrodes (Roitbak, 1953a).)

    In preparation No. 37, as a result of tens of repeated

experiments (with 5-minute intervals between experiments), it was

possible to be persuaded that the infrequent P^1_{10} stimulation, at

interruption of P^2_{50} tetanization, continued for a period of 40-60

seconds to produce new effects the same in character as those that

arose at the time of the combination of P^1_{10} + P^2_{50}, positive

fluctuations of biopotential (Fig. 52, D). If P^1_{10} stimulation

when it proceeded in an isolated way at cessation of P^2_{50} was broken

off for 10-15 seconds, then at repeated switching on this stimulation

ceased to provoke new effects, which, if it were not interrupted in

its action, it would continue to produce for 20-30 seconds more. In

other words, because of the change described in the course of the

experiment, P^1_{10} stimulation lost the capacity acquired by it as

the result of combining with tetanic stimulation, the capacity to

excite neuronic elements of the deep layers of the cortex. The P^1_{10}

now produced its usual effects, negative fluctuations of potential,

somewhat abated as compared with those which were provoked prior

to a combination of stimulations (Fig. 52, E). Thus, infrequent

stimulation of the cortex sustains a state of heightened excitability

in the focus that had arisen during combination of this stimulation

with frequent tetanic stimulation of another point of the cortex.

On the above-mentioned basis it is remarkable that if

infrequent stimulations are applied to point P_1 (P^1_{10}) at interruption

of tetanization of point P_2 (P^2_{50}) which continued prior to the

arising of a rhythmical electrical activity maximal in intensity,

then it is possible to obtain those phenomena which are observed as

the result of a combination of $P^1_{10} + P^2_{50}$ stimulations. In

experiment 52, H, the P^1_{10} stimulation was applied for 280 milliseconds

after cessation of P^2_{50} tetanic stimulation. The P^1_{10} stimulation

provoked weak effects as compared with those which it caused after

combination with P^2_{50} stimulation: the first shock caused a negative

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fluctuation; the last shocks caused positive fluctuations, but these
effects in amplitude and duration considerably yield to the effects
which P^1_{10} caused after combination with P^2_{50} and, to which attention
should be turned, in these effects additional positive fluctuation
(compare Fig. 52 H and C) was lacking in these effects or was slightly
expressed.

(Legend to Fig. 53, textpage 126: Bioelectrical phenomena in
the focus of heightened excitability created in cerebral cortex. Cat
No. 24, May 20, 1950. Continuation of experiments the recordings of
which were presented in Fig. 45. On the surface of the gyrus
suprasylvius 2 pairs of stimulating electrodes were placed (P_1 and
P_2) and between them a discharge electrode E (see scheme). A - P^1_9
stimulation and annexation of P^2_{50} stimulation. B - cessation of
P^2_{50} stimulation after 14 seconds of union of $P^1_9 + P^2_{50}$ stimulations.
C - 15 seconds after B; cessation of P^1_9 stimulation. D - end of
15-second P^2_{50} stimulation and annexation of P^1_9 stimulation. E -
continuation of D. F - several seconds after E. G - scheme of
arrangement of electrodes.)

The experiment, the recordings of which are presented in Fig.
53, A-C, is a repetition of the experiment illustrated by the recordings
of Fig. 45, analyzed in detail. As a result of the combination of
stimulations $P^1_{10} + P^2_{50}$, P^1_{10} at interruption of P^2_{50} began to cause

new complex effects. After 5 minutes an experiment was set up the

recordings of which are presented in Fig. 53, D-F. Tetanic stimulation

of a second point of the cortex (P^2_{50}) was continued up to the arising

of a rhythmical electrical activity maximal in intensity; stimulation

of the first point (P^1_{10}) was begun 0.5 sec. after cessation of P^2_{50}

tetanization (Fig. 53, D). In spite of the fact that this stimulation

was applied at the time of the aftereffect, it caused no new complex

effects (compare 53, D, E, F and 53, B and C).

Thus, it is possible to conclude that the fact of union, of

coincidence at the time of the stimulations of two points of the

cortex, has importance in the sense of determining the character of

the bioelectrical effects which one of the stimulations being combined

provokes after the cessation of the other. Thus, the effect of direct

electrical stimulation of a given point of the cortex depends on the

preceding history of stimulation in a more complex sense than has

been admitted up to now: the effect of stimulation depends not only

on the previous stimulation of a given point of the cortex and not

only on the previous stimulation of other points of the cortex

(Vvedenskii, 1897), but also on the fact of a previous coincidence

in time of stimulation of a given point of the cortex by stimulation

of any other point of the cortex.

Finally, in Fig. 54 are presented recordings of an experiment

the set-up of which was the following. Two pairs of stimulating

electrodes were established on the gyrus suprasylvius at a distance

of 11 mm. from one another, and the discharge electrode was between

them. Stimulation of point P_1 at a rhythm of 8 per second (P^1_8)

during a stimulation intensity of 4 v. did not cause appreciable

bioelectrical effects at the point being discharged, E (osc. A).

At annexation of the stimulation of point P_2 at a rhythm of 100

per sec. (P^2_{100}) gradually intensifying rhythmical activity quickly

arose at the rhythm of P_1 stimulation (osc. B).

At cessation of tetanization the alternate slow fluctuation

had a considerably larger amplitude than at the time of tetanization

(osc. C). Furthermore, the slow fluctuations proceeded at a far

more infrequent rhythm, but the effects that arose were connected

with P^1_8 stimulation: each complex effect was a group of fluctuations

at a rhythm of about 50 per second and the slow fluctuation of

potential arose in response to each second shock of P^1_8 stimulation

(osc. C and D). After several seconds the effects were simplified:

each P^1_8 shock caused a double positive fluctuation (osc. E). After

several more seconds the P^1_8 stimulation began to cause such effects

as prior to union of stimulations (osc. F).

Thus, infrequent stimulation of point P_1 of the cortex caused,

at a given part being discharged, slight electrical effects which can

be considered an expression only of polarization; afterward at this

point, as the result of P^2_{100} tetanization, a focus of rhythmical

activity having arisen, the subthreshold P^1_8 stimulation became effective:

it stipulated the rhythm frequency of electrical fluctuations at time
of combination with tetanic stimulation $P^2{}_{100}$; at interruption of the
union for a long period it remained capable of provoking excitation
of certain neuronic complexes.

(Legend to Fig. 54, textpage 128: Bioelectrical phenomena of
a focus of heightened excitability created in the cerebral cortex.
Cat No. 37, Jan. 7, 1951. (The same preparation as for Fig. 52 too,
3 hours after the experiments cited there.) Stimulating electrodes
$(P_1$ and $P_2)$ and discharge (E) electrode were placed on the gyrus
suprasylvius (see scheme). A - subthreshold stimulation (4 v.)
through electrodes P_1 at a rhythm of 8 per sec. $(P^1{}_8)$ and annexation
of energetic (30 v.) stimulation through electrodes P_2 at a rhythm
of 100 per second $(P^2{}_{100})$. B - continuation of combination of
stimulations $P^1{}_8 + P^2{}_{100}$ at uninterrupted $P^1{}_8$ stimulation (moment
of cessation of tetanization is indicated by arrow) (TN: No legend
given for C.) D - direct continuation of recording C. E - continuation
10 seconds after recording D. F - after 40 seconds; cessation of $P^1{}_8$
stimulation. G - effect of intense (25 v.) $P^1{}_8$ stimulation. H -
scheme of arrangement of electrodes (Roitbak, 1953a).)

One of the most important principles of neurophysiology that
lies at the basis of the dynamics of reflex activity of the central
nervous system was established by the investigations chiefly of
Pavlov, Vvedenskii, Ukhtomskii, and Beritov. It was formulated by

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Pavlov in 1904 in the following way: "That point of the central

nervous system which at the time of a positive reflex is highly

stimulated directs toward itself weak stimulations that fall from

the outer or inner world simultaneously on other points of this

system." As known, Ukhtomskii introduced the concept of the

dominant. The phenomenon of the dominant has been described by

him in the following way: "Every time as we step up the excitability

of a certain center and it is adapted to adding up and keeping in

itself the excitation, a chance stimulation again applied will first

of all provoke reactions in it, i.e., descriptively speaking, it will

deflect its own impulses to it" (Ukhtomskii, 1926). Beritov gave a

rational explanation to the phenomenon which bore the descriptive

designation of "attraction" of excitation, having indicated that

the variability of the reflexes depends on this, that side by side

with this center on which the stimulation acts, owing to the irradi-

ation of the excitation, other centers too are excited, the

excitability of which is heightened under the influence of any

inner or outer stimulations; the activity of the latter, which

arises first, stipulates inhibition of the reaction that usually

arises at a given stimulation although the latter can also appear

(1910, 1915, 1948; Beritov, Bakuradze, and Narikashvili, 1937).

Several examples characteristic of the phenomenon being

considered will be presented below.

1) If in dog under light narcosis, shortly after stimulation of the motor point for the extensors of the fore paw (center A), stimulation is applied to the motor point of the flexors of the fore paw (center B), then it is possible to observe a paradoxical effect: no effect is observed corresponding to stimulation of B but such an effect as would continue to stimulate center A (Vvedenskii, 1897).

2) In non-narcotized dog with electrodes placed directly on thecortex, after stimulation of part of the motor area of the cortex, stimulation of the optical part provokes movement of the animal as if the stimulation were applied to the motor part (Baer, 1905).

3) At time of the reflex acts of swallowing and defecation direct electrical stimulation of the cerebral cortex that usually causes movements of the extremities does not produce them but intensifies the existing reaction, swallowing or defecation (Ukhtomskii, 1911).

4) As a result of local strychnine poisoning of the cortex it is possible to observe inversion of action of conditional stimulation. Zal'manson (1929), after working out a conditioned defensive reflex for the left rear paw at the knock of a metronome, opened the brain and poisoned with strychnine the motor portion of the right fore paw; after poisoning he began to produce by metronome an energetic reaction in the right paw.

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5) At local strychnine poisoning of the motor area of the
cortex rhythmical movements arise of the corresponding extremity;
these movements intensify at stimulation of the most various parts
of the cortex, both of the corresponding and of the other hemisphere
(Beritov, 1917).

6) If an anode of constant current is applied to any part
of a motor area of the cortex of non-narcotized rabbit, then the
earlier "indifferent" auditory and optical stimulations begin to
cause movements of the animal that prove more intense in those
groups of muscles the motor centers of which undergo polarization;
auditory stimulations continue to cause movement sometimes for a
period of several hours after breaking off the constant current
(Rusinov, 1951, 1953; Novikova, Rusinov and Semiokhina, 1952).

Ukhtomskii endeavored to ascertain those intimate processes
which lie at the base of "heightened excitability", as a result of
which a given focus of the central nervous system reacts uniformly
to uniform influences that are applied by the current medium. He
came to the conclusion that the focus possessing heightened
excitability is in a state of fixed excitation: "from this too
there is heightened excitability, which it had already prepared by
a constant excitation already present of a given focus" (1930). He
considered a concrete study of this necessary; as it accumulates

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and as it arises, this focus functions; but he was unable to
accomplish this and he was unable "by electrophysiological ocular
demonstration to change the theoretical forecast" (Ukhtomskii, 1937).

The dominant was recently examined by Ukhtomskii too as a
fixed focus of pulsating excitation. According to Golikov's ideas,
at the arising of a dominant focus excitation irradiation can occur
over an ever greater number of central cellular elements, over ever
larger cellular masses, and, together with this, concentration of the
excitation in them. The elements of excitation "that are being recruited"
in the process begin to be excited rhythmically at one tempo and
rhythm. This is a phenomenon being expressed in the reduction and
disappearance of dispersion and in lack of coordination in the periods of
excitation and has been designated "inner synchronization", in distinction
from synchronization with the rhythm of external stimuli, which was
designated "outer synchronization" (Golikov, 1950).

In the oscillographic experiments described above with tetanization
of the surface of the cortex a focus of heightened excitability arose
that evidently possessed those features which are applied to characterize
the "dominant focus". In Figs. 43 and 47, in which an oscillographic
recording was presented of the whole course of the arising of a focus
of heightened excitability, it is well seen how stimulation over a long
time remains in apparent inactivity, how then rhythmical electrical

activity gradually begins to arise, how at first fluctuations small

in amplitude arise, the rhythm of which is irregular, how progressive

increase of amplitude of the bioelectrical fluctuations occurs, and

how their regular rhythm is established and there arises finally in

developed form a limited focus of most intense rhythmical electrical

activity. Thus, in these oscillographic recordings the process of

implication in the action of an ever greater number of neuronic elements

and the process of "inner synchronization" at the focus of heightened

excitability are registered.

During oscillographic study of the focus of heightened excitability

it is seen with what facility it is possible "to impose" the rhythm:

annexation of infrequent stimulation of another point of the cortex

leads extremely quickly to this, that the rhythm being established

in the focus changes to the rhythm of the accessory stimulation (Figs. 50

and 51).

In the investigations of Rusinov the electrical activity was

recorded "of the focus of the dominant", i.e. of the part being

polarized of the motor area of the cortex, but no special changes

were discovered there. On the other hand, in these works no

explanation is given to this, in what way the anode of constant current

leads to the arising of a focus of heightened excitability.

It is necessary to think that during application to the surface

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of the cortex of an anode of constant current at a certain low of its voltage anodic polarization of the elements of the surface layers of the cortex, namely of the top dendrites of the pyramid neurons, must occur; at this time their bodies and axons should be found in a state of catelectrotonus. Goldring and O'Leary (1951) came to a like conclusion. Entirely demonstrative in this respect are the data of Burns, obtained in experiments with isolated strip of cortex: when the anode was placed on the surface of the cortex, switching on of the current caused rhythmical excitation of the neuronic elements of the deep layers of the cortex[*]. The same effect was obtained if a cathode was added to the microelectrode inserted into layers V and VI of the cortex (compare textpages 56-57). ([*]During current intensities of greater than 600 microA. the phenomenon of slow-spreading depression arose. It is characteristic that in the experiments of Rusinov and coworkers the focus of heightened excitability arose at a current intensity of 0.5 v.; at increase of the intensity above 3 v. the "pessimal" (worst) effect, as they expressed it, set in.) If at the time of subthreshold (in the sense of provocation of rhythmical excitation) anodization of the surface of the cortex a shock of electrical stimulation was applied, then rhythmical activity of the cortical neurons arose that lasted as long as the polarization continued (Burns, 1954).

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Thus, the pyramid neurons when their bodies are in a state of catelectrotonus possess heightened excitability and begin to be excited rhythmically. However, the very same thing occurs with cortical neurons under the influence of a number of comparatively frequent impulses of excitation, and certainly in natural conditions the focus of heightened excitability is created in this last way.

As we have seen, tetanic stimulation of the surface of the cortex after several seconds of action begins to excite a certain complex of intracortical neurons; infrequent stimulations of another distant point of the cortex are not capable of provoking the excitation of this complex of neurons by any continuance of their action. Thus, the first stimulation causes excitation of the "center" in question, the second stimulation, the "indifferent", is not appreciably reflected in the state of the given center. At union of these same two stimulations after a certain time the "indifferent" stimulus becomes effective: its rhythm stipulates the rhythm of excitation of the given complex of neurons; at cessation of tetanization the stimulation that earlier was indifferent is capable for a long time, measured in tens of seconds and even in minutes, of causing a most energetic excitation of a given complex of neurons. Our "indifferent" stimulation, infrequent stimulation of any point of the surface of the cortex, causes activity that spills widely over the cortex: the excitation mainly along the system of associative fibers of layer I

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spills through the cortex, producing regional excitation of the neuronic elements mainly of the surface cortical layers. Tetanic stimulation of any point of the cortex or of the white matter produces a limited focus of energetic rhythmical activity. It is possible to think that when excitation of the entire complex of cortical neurons occurs, then in certain cases the activity does not spread through the cortex but is limited to a given focus: the process of inhibition localizes this focus. Now the waves of excitation from our "indifferent" stimulation are capable of causing and supporting most energetic activity of the given focus, this stimulation ceases in regard to the given "center" to be indifferent.

As Ukhtomskii said, the dominant plays the role of the key to clarification of the mechanism of the temporary connections (Ukhtomskii, 1924). There is no doubt that at the base of the formation of temporary connections lies the arising of foci of excitation or of foci of heightened excitability in the cerebral cortex (Pavlov, 1904; see also 1908-1909, 1909, and 1912). Theoretical consideration of the mechanism of the formation of temporal connections made by Beritov is based as a whole on examination of the interaction of the foci of excitation in regard to the foci of heightened excitability in the cerebral cortex (Beritov, 1922, 1924, 1932, 1948, 1953).

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Phenomena which were observed in the oscillographic experiments
considered are finally far from a locking of connection. (Footnote:
Conditioned reflexes can be produced in conditions of ingenious
experiments on narcotized, tied animals (Kozenko, 1952; Sterling and
Miller, 1944). On non-narcotized animals the conditioned reflex is
easily developed at combination of sound stimulation with direct
electrical stimulation of the motor area of the cortex (Kriazhev,
1934) and can be formed at combination of electrical stimulations
of two cortical areas (Livanov, 1947; Kogan, 1951). These facts
speak in favor of the conclusion that the formation of conditioned
reflexes is an elementary, readily conditioned process and that
their complexity does not consist in the intricacy of the mechanism
of their formation (Pavlov, 1913a, 1917). Hence, finally, it follows
that temporary connections can be formed in the conditions of my
oscillographic experiments, during which there was association of
many unfavorable conditions.) As Ukhtomskii said, an induced
dominant is a condition for the arising of a temporary functional
connection; after strengthening this connection it becomes a result
of it, i.e. what has been realized up to now during the effect of
two factors: the unconditional and the indifferent stimulations (in
case of the production of a conditioned reflex) now is accomplished
under the influence of just one factor, the conditional stimulation,

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which is no longer haphazard but selectively excites the focus of

the former dominant (Ukhtomskii, 1926). Certainly at the base of

the formation and strengthening of the temporary connections lie

definite morphological changes of the neuronic elements, probably

in the sphere of the synaptic endings (Pavlov, 1922, 1925; Beritov,

1926, 1932).

Part II
From Russian for Dr. Wade H. Marshall

Chapter IV

Bioelectrical Potentials Arising in the Cerebral
Cortex at Peripheral Stimulations

Normally the cerebral cortex comes into an active state under

the influence of afferent impulses that arise as the result of outer

and inner stimulations falling on the receptors. Consequently, study

of bioelectrical phenomena running high in the cortex in response to

adequate stimulations of the receptors, as well as in response to

stimulations of the corresponding sensory nerves, offers special

interest.

Nevertheless, investigations were conducted in this direction

in the pre-oscillographic era, that were often carried out at a high

level in all respects except intensification of technique and recording

of cortical biocurrents. Many of the facts obtained at that time have

not lost their importance even now. The experiments were made on

narcotized animals, on animals made immobile with curare, and on non-

narcotized animals. The cortical biocurrents were discharged
bipolarwise in the majority of cases, but they were also deflected
monopolarwise (Larinov, 1899). Cortical biocurrents were studied
when the discharge electrodes were placed on the surface of the
exposed cortex, on the dura mater, and on the cranial bones (Fleischl
von Marxow, 1890; Pravdich-Neminskii, 1913).

Great precautions were undertaken for avoidance of artefacts;
in particular, it was ascertained whether the bioelectrical phenomena
observed at temporary association with heart and respiratory activity
were found (Vvedenskii, 1884). It was found that it is very easy
to obtain artefacts in experiments on animals not rendered immobile
(Pravdich-Neminskii, 1925). Many of the methods used in this respect
were afterwards omitted, and a number of electrophysiologists at the
present time have admitted errors from method in their investigations
(see Roitbak and Khechinashvili, 1952).

A survey will be presented below of the data obtained at this
time during investigation of bioelectrical phenomena in the cerebral
cortex arising under the influence of peripheral stimulations.

Caton (1875) and Danilevskii (1876) discovered that during the
action of external stimul bioelectrical reactions arise in the cerebral
cortex. It was established at that time that electrical reactions
of the cortex disappear during deep narcosis (Fleischl, 1890; Danilevskii,
1891), and it was shown by a number of experiments that these reactions

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reflect a process of excitation of the cortical nerve elements
(Kaufman, 1912; Pravdich-Neminskii, 1913). It was ascertained that
during the action of outer stimulus electrical reaction arises only
in a certain part of the cortex, for instance at stimulation of the
cutaneous nerves (they arise) in the anterior part of the cortex and
at eye exposure (they arise) in the occipital part of the cortex.
Other areas of the cortex during this remain inactive (Danilevskii,
1891; Beck, 1890). In answer to unilateral stimulation, bioelectrical
reaction arose in the majority of cases in the opposite hemisphere
(Danilevskii, 1891). Trivus (1900) observed in non-narcotized dogs,
during stimulations of the eye with light, bioelectrical effects in
the occipital region; but sometimes they were observed too in other
regions, for instance in the temporal (which he explained by the
arising of associations).

Bioelectrical effects in the cortex were discovered during
optical stimulations (Danilevskii, 1876, 1891; Fleischl, 1890; Beck,
1890; Trivus, 1900, and Kaufman, 1912) and wound stimulations (Danilevskii;
Larionov, 1899); and during electrical stimulation of the sciatic nerve
(Beck, 1890; Danilevskii, 1891; Kaufman, 1912; Pravdich-Neminskii, 1913,
1925) and of the vagus nerve, at the stimulation of which reaction arose
in the front lobe of the cortex (Danilevskii, 1891). During pain
stimulations of the trunk and during active and passive movements of

the extremities no definite effects at all were observed in the cortex,

particularly in the cortical motor areas. (Footnote: It is interesting

that Danilevskii (1891) undertook experiments by sinking the discharge

electrodes (glass tubes) deep under the cortex into the white matter

and into the subcortical nucleus. At peripheral stimulations he observed

electrical reactions similar to those which arose in the cortex. Only

now is it possible to estimate the importance of this observation.)

Danilevskii spoke of cortical lobes in which bioelectrical

reactions arise at stimulation of the corresponding receptors or nerves.

Larimnov (1899) on the basis of his galvanometric experiments came to

a conclusion on fine localization within the limits of the auditory

region and on representation in the temporal convolutions of the

various parts of the organ of Corti.

Trivus observed in experiments on non-narcotized dogs that,

at cessation of short-term (20 sec.) exposure of the eye to light,

bioelectrical reaction of the cortex continued up to two minutes,

which indicates, in his opinion, characteristics of cortical activity

that remain for a long time after relatively short stimulation.

Vvedenskii (1884), using the telephone, listened to periodical

rolls from the cerebral cortex of rabbit and dog, which he designated

as the excitation expression of the central formations. At stimulation

of the sciatic nerve these rolls disappeared, i.e. evidently Vvedenskii

observed the phenomenon of oppression of "spontaneous" electrical

activity of the cerebral cortex at stimulation of the sensory nerve.

Kaufman (1912) likewise found that stimulation of the nerves

depresses "spontaneous" activity of the cortex. This occurred

particularly in his experiments at stimulation of the vagus nerve.

Great importance was attached to the sign of the potentials

observed. Danilevskii (1891) at stimulation of one or another

sensory organ observed the arising of negative or positive

fluctuations in certain parts of the cortex. He proposed that

the difference of sign might depend on the location of the electrodes

and/or of a different physiological state of the cortex. Trivus in

experiments on non-narcotized dogs observed that at peripheral

stimulations in the cortex in complex effect arises: a negative

fluctuation with a small positive one preceding it. He made the

assumption that simple effects in the form of purely positive

fluctuations are obtained in consequence of the falling out of the

negative fluctuation. Consequently, attempts were made to analyze

the separate components of the potentials registered, but the

solution of this problem became possible only after using boosters

and oscillographs.

Pravdich-Neminskii was the first to describe by means of a

string galvanometer the electrical fluctuations generated by the

cerebral cortex. He carried out the experiments on tracheotomized

curarized dogs; one discharge electrode was placed on the frontal,

the second on the occipital part of the cortex. As known, he

described the characteristic rhythm of "spontaneous" bioelectrical

fluctuations of the cortex and at stimulation of the sciatic nerve

he recorded characteristic bioelectrical effects arising at solitary

and tetanic stimulations of the nerve. These effects consisted

of the arising of two-phase fluctuations. At first a negative, then

a positive phase arose; the effect formed with a latent period of

75 milliseconds and more. The second positive phase was of greater

length and amplitude. The negative phase might be lacking. During

the interval between solitary stimulations, less than 2 seconds,

the second effect proved highly attenuated. Tetanic stimulations

gave a picture close to that which was observed from one shock of

stimulation, but sometimes more complex effects arose (Pravdich-

Neminskii, 1913, 1925).

On the basis of the data obtained in works devoted to the study

of bioelectrical phenomena in the central nervous system, two important

theoretical aspects were apparent: 1. A study of electrical phenomena

in the cerebrum gives an opportunity to investigate those objective

material processes which are substrate to psychic phenomena (Danilevskii,

Pravdich-Neminskii). 2. The galvanometric method can serve for a study

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of the questions of localization of the areas of perception of the

cortex (Bekhterev, Tarkhanov, Larionov, and Trivus).

1. Direct Bioelectrical Reaction of the Cerebral Cortex

to Peripheral Stimulations: Primary Responses

According to contemporary oscillographic data obtained mainly in

conditions of pointed experiments at stimulation of the receptors or of

the sensory nerves from the surface of the cerebral cortex a characteristic

bioelectrical reaction is registered. In answer to single stimulation

of the receptors (sound shock, flash of light, touching the skin, etc.)

or in response to stimulation of a sensory nerve by one brief impulse

of electrical current with a latent period the duration of which depends

on different causes, a positive fluctuation of a potential 7-15

milliseconds and more in length arises. During deep narcosis reaction

is limited to peripheral stimulation with the arising of this positive

slow fluctuation of biopotential, the amplitude of which at intensification

of stimulation is increased to a certain limit.

At shallow narcosis in response to peripheral irritation a more

complex bioelectrical reaction arises: after the positive slow potential

the negative arises, after which a number of additional fluctuations

can follow, positive or negative.

With regard to the origin of the separate components of the

"primary responses", there is a whole series of opinions, often

contradictory, which will be considered later.

At the present time it is known that "primary responses" arise
in the cortex at stimulation of peripheral portions of the following
analysors.

1) Optical. During adequate stimulations of the retina (Marshall,
Talbot, and Ades, 1943, et al.) and at electrical stimulations of the
optic nerve (O'Leary and Bishop, 1938; Marshall, 1949; Chang and Kaada,
1950, et al.) primary responses were registered only in the optic area
of the cortex; in cat they were registered from the gyrus lateralis
and from the rear pole of the gyrus suprasylvius.

2) Acoustic. During adequate stimulations of the organ of
Corti (Bremer and Dow, 1939; Gershuni, 1940; Bremer, 1943, 1952; Ades,
1943; Artem'ev, 1951, et al.), at electrical stimulation of the acoustic
nerve (Ades and Brookhart, 1950; Chang, 1951b, 1953a), and at electrical
stimulation of the organ of Corti (Tunturi, 1944) primary responses were
registered in the auditory area of the cortex; in cat they were registered
in the area of the gyr. ectosylvius med. and ant. and the gyr. sylvius,
as well as in a small area of the gyr. suprasylvius ant.

3) Cutaneous. At stimulation, adequate or electrical, of the
cutaneous receptors (Adrian, 1941, 1947; Marshall, Woolsey and Bard,
1941; Scherrer and Oeconomos, 1954), and at stimulation of the compound
and cutaneous nerves (Bartley and Heinbecker, 1938; Marshall, 1941;
Marshall and coworkers, 1941; Roitbak, 1953b; Cragg, 1954) primary

responses were registered in the area of the gyr. sigmoideus post.

and in the adjoining parts of gyr. suprasylvius and gyr. ectosylvius.

However, in experiments on monkeys at stimulation of the cutaneous

branches of the sciatic nerve primary responses were registered not

only in the area behind the central groove but also in the precentral

region where they set in with a somewhat greater latent period. They

continued to be registered in the precentral region after the removal

of the postcentral area on both sides, the removal of the cerebellum,

and the removal of the precentral area of the other hemisphere. Thus,

the possibility has been excluded of thinking that the effect arising

in the precentral area is secondary, stipulated particularly by

preliminary excitation of the elements of the postcentral area (Malis,

Pribram, and Kruger, 1953; see also Gardner and Morin, 1953).

4) Vestibular. Primary responses to electrical stimulations of

the vestibular nerve are registered in cats, according to Khechinashvili

(1952, 1953), in the region of the gyr. ectosylvius med. and post. and

of the gyr. sylvius ant.; according to Kempinsky's data (1951) and the

data of Mickle and Ades (1952), they are registered from the gyr.

suprasylvius ant. and from the adjoining part of the gyr. ectosylvius

ant.

5) Motor. Primary responses arise in the cortex at stimulation

of the sensory nerves that proceed from the muscles and joints, for

instance from the nerves of the m. soleus, the lateral and medial heads

of the m. gastrocnemius, m. flexor digitorum longus, and the nerve

from the knee joint (Gardner and Noer, 1952; Gardner and Haddad, 1953).
In experiments on cats at stimulation of these nerves effects arose in
the same areas of the cortex in which effects arose at stimulation of
the cutaneous nerves of the hind extremity, i.e. in the upper part of the
gyr. sigmoideus post. (in so-called field I of the general sensitivity)
and in the region of the gyr. suprasylvius ant. and gyr. ectosylvius
ant. (in the so-called field II of the general sensitivity). The effects
arose in both hemispheres, but they were far more intense in the
contralateral hemisphere. Analogous results were obtained at stimulation
of the posterior columns of the spinal cord and of their nuclei in the
medulla oblongata, as well as during the twitchings of the separate
muscles produced by their electrical stimulation (Mickle and Ades, 1952).
In experiments on cats no attempt was made to discharge primary responses
at muscle-joint stimulations from cortical areas found anterior to the
cross-shaped sulcus. In monkeys at stimulation of the nerves proceeding
from muscles and joints direct bioelectrical effects were registered in
those areas where effects were registered at stimulations of cutaneous
nerves, i.e. in the posterior and anterior central convolutions of both
hemispheres (Malis, Pribram, and Kruger, 1953; Gardner and Morin, 1953).

6) Olfactory. At electrical stimulation of the olfactory nerve
and the olfactory bulb (Kaada, 1951, et al.) direct bioelectrical effects
were registered in certain parts of the basal and medial surface of the
cortex.

7) Taste. At electrical stimulation of the chorda tympani
in cat primary responses were registered from a small zone, an
area 5-10 mm^2 in size, on the orbital surface of the cortex anterior
to the area of the cutaneous projection of the face. This zone over-
laps the zone of the tactile representation of the tongue (Parron
and Amassian, 1952). These oscillographic data confirm the opinion
of Bekhterev on this, that in the cortex the area of taste perception
must be found there where the motor parts of swallowing and chewing lie,
and they also confirm the results of a study of war injuries and of
experiments on monkeys, according to which the taste area overlaps
part of the general sensitivity of the tongue (see Beritov, 1948).

8) Visceral. At electrical stimulation of the n. splanchnicus in
cat primary responses were registered in two regions, in the gyr.
sigmoideus post. and in the anterior part of the gyr. ectosylvius ant.
i.e. in fields I and II of the cutaneous projection region at the edge
of portions of the front and rear paw (Amassian, 1951). Downman (1951)
localizes the second area, where primary responses are deflected at
stimulation of the n. splanchnicus, in the region of the lower part
of the cutaneous representation of the face. It should be noted that
on the basis of experiments with stimulation of the exposed cortex
Rasmussen and Penfield (1947) find that in man the cortical field
connected with the sensation of the organs of the abdominal cavity is

located at the lower end of the cutaneous projection region. At
mechanical stimulation of the intestines and stomach of rabbit an
arising of bioelectrical reactions was observed in the posterior
portion of field 8, the anterior pariental, the postcentral, the
orbital, and the retrosplenial regions of the cortex (Lisitsa,
1941).

At stimulation of the vagus nerve intensification of electrical
activity in the orbital region of the cerebral cortex of cat (Bailey
and Bremer, 1938) was ascertained, i.e. the results of oscillographic
experiments confirmed the fact established by Danilevskii in 1891
(see above).

Thus, at stimulation of the peripheral end of any analysor
in the cerebral cortex a characteristic bioelectrical reaction (primary
response) arises. In other words, during different peripheral
stimulations of the surface of the cortex effects similar in character
are discharged. The identical character of the cortical bioelectrical
reaction in response to afferent impulses most diverse in origin
indicates the identical method of termination of all afferent fibers
in the cortex.

Primary responses to stimulation of receptors according to their
temporal course and form differ somewhat from responses provoked by
stimulation of the corresponding nerves. This is explained finally by

the fact that there proceed in the central nervous system discharges
of afferent impulses different in character. It is known, for example,
that a discharge of impulses arising at stretching a tendon, i.e.
a most synchronous discharge of impulses from natural stimulation,
is much less synchronous than a discharge at electrical stimulation
of a nerve (Barron and Matthews, 1938).

At a frequency of peripheral stimulations of 1-50 per second,
particularly of shocks of sound, "primary responses" follow the
rhythm of the stimulation. In connection with increase of frequency
of stimulation, reduction of amplitude and change in the character
of responses are observed. During the effect of musical tones a
bioelectrical potential arises first in the auditory projection
region, similar to that which is provoked by one sound shock, and
then, during the continuing effect of the tone, intensified rhythm
is observed of quick fluctuations (Bremer, 1943), or the formation
is observed of slow potentials rhythmically following one another
(Tunturi, 1949).

(Legend to Fig. 55, textpage 139: A - convolutions of the
dorso-lateral cerebral surface of cat. 1 - splenialis, 2 -
sigmoidalis (cruciatus) ant., 3 - sigmoidalis (cruciatus) post.,
4 - coronalis, 5 - suprasylvius med., 6 - suprasylvius ant., 7 -
suprasylvius post., 8 - ectosylvius med., 9 - ectosylvius ant.,

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10 - ectosylvius post., 11 - sylvius ant., 12 - sylvius post., 13 -

orbitalis (according to the Atlas of the Cerebrum of Man and of

Animals of the Institute of the Brain, M(oscow), 1937, and according

to Kaada, 1951). B - Localization of visual (I-top center and II-

top right), auditory (I-center and II-lower right), and cutaneous

(I-upper and II-lower left) of the projection regions of the cerebral

cortex of cat, determined on the basis of oscillographic data (Rose

and Woolsey, 1949). C - Region of Cerebral cortex of cat from which

bioelectrical potentials are registered at stimulation of the pyra-

midal pathways; by meshing the portions are noted in which potentials

of greatest amplitude arise (Woolsey and Chang, 1948). D - Regions of

the cortex, in which primary responses arise at stimulation of the

n. splanchnicus (hatched) and of the chorda tympani (dotted) (according

to Amassian, 1951; Downman, 1951: Patton and Amassian, 1952).)

In Fig. 55 maps are presented of the cerebral cortex of cat;

certain projection areas are plotted on them, determined on the

basis of oscillographic experiments (B and D - the two at the right).

The area of the cortex from which bioelectrical effects are registered

at stimulation of the pyramidal pathways, i.e. at antidromic excitation

of the neurons that produce a beginning to pyramidal pathways, is

presented separately. In other words, Fig. 55 C (lower left), shows

the cortical territory where the main mass of pyramidal neurons is

found, the axons of which take part in the formation of the pyramidal pathways. As seen, this territory occupies a region of the cortex anterior to the crosswise sulcus, as well as the region of the cutaneous-muscular projection. Betz cells are arranged most closely in the region anterior to the gyr. cruciatus, as well as in the region of the gyr. ectosylvius ant. (2 and 9) (i.e. in the region of the so-called field II of general sensitivity). (See also Lance and Manning, 1954, and Porter, 1955.)

It is known that stimulation of different parts of the motor region of the cortex in animal provokes movement of the different parts of the body: the upper part of the motor region of the cortex is linked through segment L_3-S_2 of the spinal cord with the muscles of the rear extremities, the middle part is linked with the muscles of the front extremities, and the lowest part with muscles of the muzzle and head, i.e. the Betz cells, connected with muscles of different parts of the body, are arranged in a certain order along the strips of cortex. It is known that relationships likewise exist between points of the cortex and points of the surface of the skin: impulses from the skin on the muzzle and head lead to the lower parts, from the skin on the front extremities to the middle parts, and from the skin on the hind extremities to the upper parts of the cutaneous projection region of the cortex. This was ascertained

on the basis of a study of the activity of the cortex by a method

of conditioned reflexes (see Pavlov, 1926), of an analysis of the

symptoms of local brain injury in people (see Adrian, 1947), of

experiments with local strychnine poisoning of the cortex (see

Beritov, 1948), and finally of the results of direct electrical

stimulation of the cerebral cortex in people at the time of dis-

section of the brain under local anesthesia in connection with

epilepsy (Penfield and Rasmussen, 1950). The method of oscillographic

registration of indirect bioelectrical effects provoked by peripheral

stimulation gave an opportunity to clarify this question extremely

accurately. Contact with several fibers in cat causes at a corres-

ponding point of the cortex a primary response of sufficient

amplitude to be registered, and thus it is possible to transpose

all points of the cutaneous surface to the cerebrum, i.e. to

establish projection in the cortex of the cutaneous receptors (Fig.

56, textpage 140: Cutaneous projection region in the cerebral cortex

of cat, ascertained by the oscillographic method in experiments on

narcotized animals. (According to Adrian, 1941)). It was found

that in addition to the classical cutaneous projection region (field

I of general sensitivity) there is a supplementary region (field II),

where the perceptive portions of the different parts of the body

overlap one another (Fig. 55, B). It was also found that the territory

of cortical projection of a given part of the body depends on the role

which in the course of evolution this part of the body has acquired in

the animal in question. For example, in monkey the cortical territory

of an upper extremity is larger than the territory of the entire trunk;

in pig (probably guinea pig is meant) the cortical territory correspond-

ing to the projection of the skin of the muzzle is greater than the

territory of projection of all the rest of the body (Adrian, 1947).

The perceptual region for muscle-joint stimulations overlaps to

a considerable measure the cutaneous perceptual region. In the cortex

there is a projection of the muscle-joint receptors similar to the

cutaneous projection. This was established by Krasnogorskii by means

of a method of conditioned reflexes and this has been confirmed by

the latest oscillographic investigations (Malis and coworkers, 1953).

With regard to the organization of the auditory projection

territory of the cortex the following is known. As said, Larionov,

on the basis of experiments with partial extirpations of the temporal

lobe of the cortex in dog and on the basis of a study of the bioelectrical

reactions arising here at sound stimulations, tried to demonstrate that

various musical tones are perceived by different parts of the cortex and

that these cortical parts are located in a certain order according to

the height of the tones perceived by them. Furthermore, at study of the

phenomenon of extinction of conditioned auditory reflexes it was concluded

that in cerebral cortex of dog there is projection of the organ of

Corti (Ivanov-Smolenskii, 1924). Tunturi, using the method of

oscillographic registration of primary responses, found that in

dog the territory of the gyr. ectosylvius med. (representing almost

the same long strip of cortex as the strip of the cutaneous projection

territory) is linked with different parts of the membr. basilaris

in such way that the high tones (8000-16,000 hertz) provoke primary

responses in the anterior part of the convolution, the lower tones

(100-400 hertz) provoke them in the posterior part, successive

octaves being represented in the cortex by 2-millimeter sections

of the convolution (Tunturi, 1944). During the effect of a given

tone a focus of highest bioelectrical activity arises, surrounded

by territory from which potentials of lesser amplitude are registered.

Furthermore, there are additional territories: in the gyr. ectosylvius

ant. bioelectrical reactions arise only in response to lower sound,

and in the gyr. ectosylvius post. reactions arise in response to

all sounds, but only during their greater intensity, and localization

of sounds is lacking here (Tunturi, 1950).

In experiments with conditioned reflexes it was found that

at the effect of a musical tone excitation seizes the territory of

the sound analysor at 3 octaves above and 1.5 octaves below the tone

being tested (Andreev and Mutli, 1938). At comparison with oscillographic

data, it can be concluded that in the gyrus ectosylvius med. neuronic
elements come into excitation for a part about 9 mm. in length and that,
according to the measure of withdrawal from the focus of greatest
activity, an ever smaller number of excited elements appear.

In cats there has been success in establishing localizations
of tones within the auditory territory: its various portions react
identically to different tones, it being impossible to explain this
phenomenon by irradiation of the excitation to the whole auditory
territory at excitation of any part of it (Bremer, 1943). At further
investigation of this problem it was successfully discovered only
that the lower and anterior parts of the auditory territory perceive
tones lower than the upper and posterior parts do (Hind, 1953). At
investigation of the biopotentials of the auditory cortex of cat by
means of microelectrodes it has been shown that the separate cell in
question from the auditory territory gives maximal discharge at the
effect of a certain tone and gives a lesser effect in response to
adjacent tones. In the latter case the discharge sets in with a
greater latent period and contains a smaller number of impulses
(Thomas, 1952). Thus, it is necessary to assume that in cat in the
auditory territory there is no strict localization of neurons in the
sense of a lineal arrangement corresponding to the sound scale and
that neurons perceiving the different tones are intermixed with one

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another. At the effect of a certain tone mainly excitation occurs

of certain neurons which are excited in response to the effect of

adjacent tones with lesser intensity, and in response to the effect

of distant tones no excitation at all is provoked. (See also Davies

and coworkers, 1954).

The more precise determination made recently of the territory

of the discharge of "primary responses" to sound stimuli have shown

that the area of the sound projection territory in cat is considerably

greater than this was at first established: the territory ascertained

by Bremer includes the gyrus ectosylvius med. and the adjacent parts

of the gyrus ectosylvius ant. and gyrus sylvius. The territory

established by Mickle and Ades (1953) embraces the anterior, middle,

and, in part, the posterior ectosylvius convolutions, the entire

gyrus sylvius, and the lower part of the gyr. suprasylvius ant. (Perl

and Casby, 1954, employing an unusual method of deflection of biopotentials,

came to the conclusion that the area of the auditory territory of cat

is considerably smaller.) Potentials of the greatest amplitude were

discharged from the territory established by Bremer; from additional

territories potentials of considerably lesser amplitude and of a

somewhat different character were discharged. In monkeys Pribram and

coworkers (1954) likewise discovered that the territory of deflection

of primary responses to sound stimulations was considerably greater

than this was established earlier and embraces the temporal and parietal

territories of the cortex as a whole. However, potentials of the

greatest amplitude were discharged from the area established earlier

and quite variable effects of lesser amplitude were discharged from

additional territories.

Thus, oscillographic data obtained at study of a sound analysor

in cat confirm Pavlov's conclusion that the number of perceptual

neurons is greatest at the center of the analysor and constantly

decreases in the measure of removal from center to periphery.

In cat from the portion of the cortex in the territory of the

lower parts of the gyr. ectosylvius ant. and gyr. suprasylvius ant. direct

bioelectrical reactions are discharged in response to vestibular, sound,

and proprioceptive stimulations (Mickle and Ades, 1952). However, this

same portion produces primary responses also to stimulation of any

portion of the skin, since here part of field II is found of the

cutaneous projection territory. It is possible also to conclude that

this portion is excited first even at stimulation of the n. splanchnicus,

since the second projection zone for the fibers of this nerve is found

in the region of the cortex. Thus, in cat in the cortical region being

considered sound, vestibular, proprioceptive, and cutaneous receptors

are represented and perhaps the receptors of the internal organs.

Evidently this circumstance has a certain physiological importance.

(Perl and Casby (1954) also observed the arising of primary responses
to sound shocks in a portion of field II of the general sensitivity.
They noted the fact of the reciprocity of these potentials and of
potentials produced here at stimulation of the contralateral sciatic
nerve.) It is interesting to note that in this territory very densely
arranged pyramidal neurons produce the beginning to the pyramidal
pathways.

Recently in the cerebral cortex of cat several of the portions
were oscillographically determined in which in response to peripheral
stimulations definite bioelectrical potentials arise in consequence of
the transmission here of impulses of excitation from the initially excited
territories of the cortex, namely from the auditory projection territory
to the lower part of the posterior ectosylvius convolution (Ades, 1943;
Bremer, 1952), from the optical and auditory projection territories
to the portion of the cortex within the suprasylvius fissure (Marshall
and coworkers, 1943; Clare and Bishop, 1954), from the cutaneous optic,
and auditory projection territories to the anterior part of the lateral
convolution (Amassian, 1954). Finally, there are data that in the
portion of the cortex behind the gyrus sigmoideus post, are registered
very irregular effects at stimulation of the n. splanchnicus (Newman,
1952). On the basis of experiments with cross sections of cortex it
is possible to conclude that transmission of excitation from the

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projection territories to other parts of the cortex is realized both

by means of a system of fibers that proceed in the cortex proper and by

means of association fibers proceeding in the white matter under the

cortex (Ades, 1943; Bremer and coworkers, 1954). However, not all

of the facts just cited can be considered firmly established. There

is still no precise knowledge concerning the reciprocity of bioelectrical

effects. According to the data of Marshall and coworkers (1943),

potentials that arise in the part of the cortex observed by them at

the effect of sound and at the effect of light do not appreciably

influence one another.

The projection territories of the different receptors in the

cortex, established by means of the oscillographic method, correspond

to definite cytoarchitectonic fields of the cortex[x] and apparently

correspond to territories in which, on the basis of experiments with

a combination of the method of conditioned reflexes and with

extirpations of one or another portion of the cortex, the nuclei of

the cortical analysors were localized. ([x]Visual projection territory

in the cortex of cat and rabbit, established on the basis of oscillo-

graphic investigations, agrees precisely with the territory of the

cortex which was designated visual territory in these animals on the

basis of morphological investigations (O'Leary and Bishop, 1938).

With regard to the auditory territory, this question still cannot be

considered clarified. The sound projection territory of cat,

established oscillographically by Bremer, corresponds to a certain

cytoarchitectonic field (Bremer, 1943). However, on the other hand,

this cytoarchitectonic field occupies only part of the territory

where Mickle and Ades (1953) registered "primary responses" to sound

stimulations. Tunturi's data (1950) with regard to the site of

auditory projection territory in dog far from agree with the results

of Mering's morphological investigations (1952): according to

Tunturi, auditory projection occupies the posterior, middle, and,

in part, the anterior ectosylvius convolutions: according to Mering,

it occupies the posterior and middle ectosylvius convolutions, as

well as the posterior and middle suprasylvius convolutions.)

In cat at bilateral removal of the auditory projection territories

no marked changes of the sound-conditioned reflexes occur. Nevertheless,

at removal of parts of the cortex including, besides auditory territories

I and II, the gyr. ectosylvius post. and field II of general sensitivity

(see Fig. 55), acute derangement of the reflexes conditioned to sound

occurs (Meyer and Woolsey, 1952). It should be noted that the territory

of the cortex removed in this case included exactly that territory where

Mickle and Ades deflected primary responses to sound.

After thermocoagulation of the auditory projection territories

in both hemispheres the animal lost the capacity to be aroused by sound

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stimulations, whereas under the influence of cutaneous stimulations

the animal aroused immediately (Bremer and Terzuolo, 1952). At

removal of cutaneous projection territories I and II in cat the

differentiation of cutaneous stimulations is greatly disturbed (Zubek,

1952).

Thus, a study of the primary cortical responses to peripheral

stimulations acquires special interest, inasmuch as it pursues the

aim of clarifying the arrangement and activity processes of the

analysors, in which the nerve impulses proceeding along the afferent

fibers stipulate the arising of depressions where the higher analysis

occurs which determines the equilibration of the organism with the

outer medium (see Andreev, 1952).

We have seen that oscillographic investigations have confirmed a

series of facts established by the method of conditioned reflexes.

Scores of further examples can be added to this. On the basis of a

study of cutaneous conditioned reflexes provoked at symmetrical points

of the right and left sides of the cutical surface, Bykov (1924) and

Bykov and Speranskii (1924) concluded that symmetrically located

points of both hemispheres are simply connected with one another in

a functional respect. This is confirmed by an oscillographic study

of this question (Curtis, 1940; Chang, 1953). Pavlov concluded that

the afferent fibers of muscle-tendon sensation terminate in the precentral

and postcentral convolutions (Pavlovskie spedy, I, p. 225). This

was oscillographically confirmed only recently (Malis and coworkers,

1953; Gardner and Morin, 1953). Andreev (1924), at studying con-

ditioned reflexes in dogs with partially injured cochleas, demonstrated

the accuracy of Helmholtz's theory. Now this has been demonstrated on

the basis of a study of the biocurrents in the separate auditory

fibers (Tasaki, 1954) and of the cortical biopotentials arising in

response to stimulation of the various portions of the membr. basilaris

(Tunturi, 1944). Zelenyi and Rukoviatkina (1925) demonstrated that

the auditory cochlea is connected with both hemispheres. This is

confirmed by the data of oscillographic investigations (Bremer, 1943;

Mickle and Ades, 1952). (However, in the territory of the gyrus

suprasylvius ant. a special portion is observed in which primary

responses arise only at stimulation of the contralateral organ of

Corti (Merlis and Lombroso, 1955).).

The agreement of the data obtained by the method of conditioned

reflexes and at study of bioelectrical phenomena in the cortex does

not disparage, finally, the importance of oscillography, but only

confirms the value of this method of investigation.

The main purpose of the experiments, the results of which will

be stated below, was clarification of the origin of bioelectrical

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potentials arising in the cortex at peripheral stimulations.

Variations of tests were used for this, similar to those which

were used for clarification of the origin of the biopotentials

arising at direct stimulation of the cortex, i.e. changes were

studied of the biopotentials in connection with changes of

frequency, intensity, and length of peripheral stimulations and

in connection with strychnine poisoning. Experiments were set

up with deflection of biopotentials from different layers of cortex,

etc. For interpretation of the facts obtained, data were used on

the structure of the cortex and an attempt was made to refer

definite components of the bioelectrical reactions of the cortex

to the activity of one or another set of neuronic elements. The

bioelectrical phenomena were studied that arise at corresponding

peripheral stimulations in cutaneous and aditory analysors of the

cortex.

Direct bioelectrical effects arising in the cutaneous analysor

at corresponding peripheral stimulations. In the majority of these

experiments the central end of the sectioned sciatic nerve was

stimulated by electrical stimuli 0.2 millisecond in length. In a

number of experiments the skin was subjected to electrical stimulation.

The following method of stimulation was used: to a certain portion of

the skin at a distance of 0.5-1 cm. from one another plate electrodes

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were attached covered with a paste of clay kneaded in a saturation

solution of calcium chloride; stimulation was made with stimuli

0.5 millisec. in length. With such a method of stimulation of the

skin the thresholds of provocation of the bioelectrical effects in

the cortex as well as the thresholds of provocation of the reflex

movement of the extremity is comparatively low: cortical bioelectrical

reactions were recorded at an intensity of the stimulating current

of the order of 2-3 volts.

If the discharge electrode is placed on the surface of the

cortex in the region of the upper part of the gyr. sigmoideus post.,

i.e. in the territory of the projection of the skin of the rear pay,

then at stimulation of the sciatic nerve of the opposite side it is

possible to register bioelectrical potentials the character of which

changes depending on the depth of the narcosis, on the intensity,

the frequency and the length of the stimulation, on the condition

of the cortex, etc.

The effect of a single stimulation. At application to the

sciatic nerve of deeply narcotized cat of a single shock of electrical

stimulation of intensity sufficient for provocation of A waves, from

the surface of the cortex in the region indicated above the following

electrical effect is registered: 7-10 milliseconds after application

of stimulation a positive fluctuation of the bioelectrical potential

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arises about 10 milliseconds in length. In some cases effects can

be recorded, in which the slow positive potential preceded somewhat

the very quick fluctuations of the common length of about 2-3 milli-

seconds (Fig. 57, A). The amplitude of the positive slow potential

usually did not exceed 0.1-0.2 milliv., i.e. the amplitude of the

initial positive potentials discharged from the surface of the cortex,

even at energetic stimulations of the sciatic nerve, was many times

less than the amplitude of the positive biopotentials arising at

direct stimulation of the cortex. As seen in Fig. 57, A, the positive

slow potential arises without marked pause after the afferent impulses;

it is possible to come to the same conclusion at examination of

recordings of effects of the optic territory of the cortex that arise

during stimulation of the optic nerve (Marshall and coworkers, 1943; Chang

and Kaada, 1950; Marshall, 1949). Thus, the latent period of the

arising of the positive slow potential registered from the surface of

the cortex after application of the peripheral stimulation is determined

at that time which is required for afferent impulses to reach the

cortex. Evidently this potential forms directly under the influence of

the afferent impulses.

(Legend to Fig. 57, textpage 146: Characteristic bioelectrical

potentials discharged from the surface of the gyr. sigmoideus post.

(region of the projection of the skin of the rear paw) at single

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stimulations of the sciatic nerve of the opposite side. A - cat.
No. 13, Dec. 16, 1949; deep narcosis. B, C, and E (the bottom three) -
narcosis of medium depth; B and C (the lower-left two) - cat No. 32,
July 10, 1950; E (lower right) Cat No. 13. D (upper right) - light nar-
coris, cat No. 43, June 5, 1951.)

At release of animal from narcosis the bioelectrical reactions
in response to peripheral stimulations became more complex. Most
often, after the positive fluctuation considered above, a negative
fluctuation sets in (Fig. 57, B). The positive fluctuation without
pause passes over into the negative, i.e. a two-phase potential
arises. The negative fluctuation of potential is quite variable in
its amplitude (it can be less or more than the initial positive one),
in its length (in the majority of cases it lasts longer than the
initial positive potential), and in its configuration.

At very light narcosis and with a good functional state of the
cortex it is possible to record complex and diverse effects.

a) After a negative potential it is possible to trace a further
series of irregular fluctuations of bioelectrical potential. Such
effects were recorded by Marshall, Woolsey, and Bard (1941) for monkeys
almost released from narcosis, in response to tactile stimulations of
the skin.

b) After a negative potential it is possible for a prolonged
negative fluctuation of the A-wave type to set in (Fig. 57, C (lower

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left). Similar effects were registered in the optic region of the

cortex of rabbit in response to stimulation of the optic nerve (O'Leary

and Bishop, 1938).

c) After a negative potential it is possible for a positive

potential of greater amplitude to arise (Fig. 57, D (upper right)).

d) In certain cases about 4 milliseconds after the beginning

of the positive potential an intense negative potential arose

approximately 5 milliseconds in length, after which a positive

fluctuation followed that shifted then into a negative (Fig. 57, E

(lower right)). This complex effect arose in certain experiments

only at intense stimulations, were as at stimulations of less

intensity the relatively simple two-phase potential described arose.

Effect of stimulations at a rhythm of 5-50 per second. The

different character of the effect of separate shocks of stimulation

of the sciatic nerve in different preparations can be connected with

the different depth of the narcosis. However, the variability of the

effects can demonstratively protrude in experiments on one preparation

at repeated stimulations of the nerve at a frequency of 2-10 per

second. For example, in the experiment the recordings of which are

presented in Fig. 58, C and D (the lower two), in the course of 1

second of stimulation it was possible to observe almost all variants

of the bioelectrical reactions enumerated above. As we shall see

below, this variability of cortical response potentials is mainly

the result of the functional state changing from moment to moment of

neuronic elements of the cortex and not of elements of subjacent

portions of the central nervous system - of the spinal cord or

thalamus, i.e. this alterability evidently depends not on the fact

that from one shock of stimulation to another the afferent "impulsation"

is changed in the sense of the increase or reduction of the number of

impulses proceeding into the cortex or in the sense of the increase

or reduction of the synchronous character of these impulses.

As said, at the time of deep narcosis reaction to stimulation

of the sciatic nerve is limited by the arising of positive potentials,

which arise stereotypewise at each shock of stimulation; the amplitude,

length, and configuration of these positive potentials are comparatively

constant. However, at light narcosis, when additional variable

bioelectrical fluctuations arise, the initial positive fluctuations

become variable, a very interesting circumstance to which special

attention has not yet been turned. In Fig. 58, C and D, it is seen

that when the effect at stimulation of a nerve is limited by a positive

potential, then the latter has a relatively greater amplitude and length

(for instance, the effects at shocks 1 and 9 of stimulation, recording

D; at shock 6, recording C). When after the initial positive potential

additional fluctuations arise, then its amplitude and length are greatly

reduced (see effects at stimulation shocks 2, 4, 6, and 8, recording D).

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In certain cases an initial quick positive fluctuation arises which
can be considered a potential of axonic origin, and after it an
energetic negative potential sets in (the effect at shock 5 of
stimulation, recording C); at examination of such effects it is
possible for the impression to be created that the positive potential
generally does not arise, but that after the advent of afferent
impulses, as the first cortical reaction, a negative potential arises.

(Legend to Fig. 58, textpage 148: Alterability of nerve
responses at a frequency of stimulation of the sciatic nerve of
5-10 per second. A - cat No. 13; B - cat No. 32; C and D - cat No. 43.)

(Legend to Fig. 59, textpage 149: Bioelectrical potentials of
the skin-receptor region of the cortex, arising at stimulation of the
skin. Cat No. 60, Nov. 5, 1953. Nembutal. Cortical potentials are
discharged through the dura mater; the discharge electrode is placed
on the region of the projection for the skin of the front paw. The
skin of the lower part of the opposite fore paw is stimulated. Intensity
of stimulation 4 v., frequency 10 per second. A - beginning of
stimulation. B - duration. C - effect of the same stimulation at a
lesser rate of exposure.)

In Fig. 59 bioelectrical potentials are presented that are
registered at stimulation of the skin on the paw. As seen, in the
cortex bioelectrical potentials thereby arise of the same character as

at stimulation of the sciatic nerve. The latent period of their

arising equals 5.5-6 milliseconds, i.e. it is shorter than in case

of stimulation of the sciatic nerve, which can be explained by the

lesser distance from the place of stimulation to the place of

discharge. The positive potentials has a length of 13-18 milli-

seconds (see osc. A, effect at the first shock of stimulation).

The greater length of the initial positive potential can be explained

by the fact that in case of stimulation of the skin receptors in

the cortex a less synchronous, i.e. a more prolonged, discharge of

afferent impulses comes to the cortex (compare Roitbak, 1950). In

the course of stimulation at a rhythm of 10 per second the effects

change from one shock of stimulation to another (osc. C). The first

3 shocks of stimulation cause simple effects, positive potentials

of considerable amplitude and length, then each shock of stimulation

begins to produce more complex effects: after the initial positive

potential a negative arises of considerable amplitude and length;

thereby the length and amplitude of the initial positive potentials

are reduced; finally, certain effects consist of the +-complex, after

which a positive fluctuation of greater amplitude follows (compare

with Fig. 57, D).

On the basis of the recordings of Figs. 58 and 59 it is

possible to make the following factual conclusion: the more powerful

the negative potential after the initial positive one, then the weaker
and shorter is the latter. In some cases it can amount to a quick
potential in the character of a peak. The positive potential has a
maximal length and amplitude in those cases when additional fluctuations
of potential set in after it. However, these relationships are not
normal; intensified negative potentials can be associated with
intensified initial positive potentials.

In certain cases the first shock of stimulation causes a complex
effect (as in Fig. 57, E), and already in response to the second shock
of stimulation at a frequency of 10 per second the effect becomes
simpler: the quick negative fluctuation that interrupts the positive
slow potential (Fig. 61, A) falls out.

At a stimulation frequency of 50 per second an effect arises
similar to the effect at one shock of stimulation, and after its
completion subsequent shocks provoke highly attenuated fluctuations
(Fig. 60, textpage 150: Bioelectrical reaction of the cortex at
tetanization of the sciatic nerve. Cat No. 13. A - beginning; B -
end of stimulation of the sciatic nerve of the opposite side;
frequency of stimulation 50 per second; end of stimulation indicated
by arrow.). It is characteristic that so long as the effect lasts
that was caused by the first shock of stimulation the shocks of
stimulation falling at this time do not produce corresponding fluctuations

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of potential. At the time of tetanic stimulation of the sciatic

nerve intensified slow fluctuations of potential arise. In this

case the fluctuations of potential that set in at the rhythm of

the stimulation are arranged on a background of slow fluctuations

of various intensity and length. After brief tetanic stimulation

of the nerve it is possible to observe intensification of the

"spontaneous" electrical activity.

Change of cortical response potentials at prolonged stimulations.

At long stimulations (more than 1 minute) at a rhythm of 10 per second

it is possible to observe that the effects of stimulation become

simpler because of the falling out of certain components of the

bioelectrical reaction. For instance, in the experiment the recordings

of which are presented in Fig. 61 at the start of stimulation after an

initial positive potential, as a rule, a negative potential arose;

after 1.5 minutes of stimulation the two-phase effects began to

alternate with the one-phase. Thus, prolonged stimulations are

reflected mainly in an additional negative fluctuation, following

after the initial positive one, which is extremely stable: positive

potentials continue to arise after several minutes of stimulation at

a rhythm of 10 per second and nowhere do they dwindle to nothing,

although at deep narcosis they quickly and considerably attenuate.

Likewise, even at a frequency of 50 per second during prolonged

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stimulations fluctuations continued to arise at the rhythm of

stimulation.

(Legends to Figs. on textpage 151, Fig. 61: Change of

bioelectrical reactions of the cortex at prolonged stimulation

of the sciatic nerve. Cat No. 13. A - beginning of stimulation

of the sciatic nerve of the opposite side; frequency of about

14 per second; B - after 1.5 minutes of stimulation. Fig. 62:

Local character of initial responses. Cat No. 43. Bioelectrical

potentials are discharged simultaneously from the upper part of

the gyrus sigmoideus post. (upper curve) and from the anterior

part of the gyr. suprasylvius. The sciatic nerve of the opposite

side is stimulated. Intensity of stimulation 1.5 v.; frequency

15 per second.)

Region of discharge of the cortical response biopotentials

at stimulation of the sciatic nerve. In experiments on narcotized

animals, corresponding with literary data, it was demonstrated

that these effects are very local; i.e. they arise only in a certain

region of the cortex corresponding to the projection of the hind

paw (in the upper part of the gyr. sigmoid post.). If an electrode

is changed to another position several millimeters lower, to a region

corresponding to the projection of the trunk or of the fore paw,

direct effects of stimulation of the sciatic nerve cease to be discharged.

In Fig. 62 recordings of the experiment are shown in which potentials
were discharged simultaneously from the upper part of the gyrus
sigmoideus post. and from the anterior pole of the gyrus suprasylvius.
As seen, at stimulation of the sciatic nerve initial responses arose
only in the region of the skin analysor. Thus, stimulation of the
sciatic nerve produces primary responses in a limited region of the
cortex and does not produce them in other regions, even those located
very nearby. The potentials that arose do not spread to other parts
of the cortex, as this is observed at direct stimulation of the
cortical surface, where punctate stimulation of the surface produces
bioelectrical response potentials over a wide territory.

Thus, we again encounter an extraordinary local quality of
bioelectrical phenomena in the cortex: the bioelectrical potentials
that arise are not spread physically for any considerable distance;
the territory of their response actually consists of the territory
of their arising. This important principle, which was proved in the
preceding chapter, oscillographic study of the cortex makes perspective.
If it should be, as proposed by certain electrophysiologists, that
the bioelectrical potentials spread diffusely through the brain and
the biopotential being registered can have an origin most diverse
in the sense of its place of generation, origin, then rational
analysis of the recordings obtained would be impossible.

Effect of strychnine poisoning. In Fig. 63 typical

oscillograms are presented, showing the effect of strychnine on

potentials arising in the cortex at single stimulations of the

sciatic nerve. (Legend to Fig. 63, textpage 153: Effect of

strychnine on primary responses arising at single stimulations

of the sciatic nerve. A - cat No. 13; the shock of stimulation is

applied to the sciatic nerve of the opposite side. After this,

local strychnine poisoning was made (0.1%) of the cortex under

the discharge electrode. B - effect of the same stimulation 2

minutes after poisoning. C - cat No. 32; effect of single stimula-

tion of the sciatic nerve (2 volts). D - Effect of the same

stimulation 3 minutes after local strychnine poisoning (1% solution)

of the cortex under a discharge electrode.) In recording A is

shown a biopotential registered from a given point of the gyrus

sigmoideus before poisoning; the latent period of its arising equals

8 milliseconds; the positive phase has an amplitude of 240 microvolts

and lasts 8 milliseconds. The negative phase lasts approximately 30

milliseconds, and its amplitude equals 150 microvolts. Local strychnine

poisoning with a 0.1% solution of strychnine was then made of the

portion of the cortex under the discharge electrode. 90 seconds after

poisoning the effect of the same stimulation of the sciatic nerve was

recorded (recording B). As seen, the effect was extraordinarily

intensified: the positive and negative phases reached amplitudes of
750 microvolts and 1300 microvolts, respectively, i.e. as a result of
the poisoning by strychnine the positive phase was increased approximately
3 times and the negative was increased more than 10 times; after the
negative phase a second positive phase now arises (about 700 microvolts).
The latent period of the effect after poisoning of the cortex by
strychnine was not changed.

A like experiment was made on another preparation (recordings
C and D). Poisoning was made with a 1% solution of strychnine. As
a result of the poisoning an increase of amplitude of the first
positive potential (from 90 to 120 microvolts) and of the negative
potential (from 75 to 600 microvolts) occurred. In the case in
question intensification of biopotentials did not reach such a degree
as at poisoning with a 0.1% solution of strychnine on another preparation.
As already said, strychnine in greater concentration can show a slight
stimulating effect and even lead to depression of bioelectrical
activity.

Thus, under the effect of strychnine extraordinary intensification
occurs (more than 10 times) of the negative phase and a certain intensifica-
tion of the positive phase of the primary responses. The effects are
graduated, depending on t he energy of stimulating of the nerve. For
instance, several minutes after the recording shown in Fig. 63, B,

stimulation of the sciatic nerve (1.5 volts) caused an effect identical
in character; the amplitude of the positive phase equaled 420 microvolts,
that of the negative phase 1.2 microvolts; at a stimulation intensity of
15 volts, the amplitude of the positive phase increased to 600 microvolts,
the negative to more than 1.5 millivolts.

At a nerve stimulation frequency of 10 per second, a peculiar
phenomenon is observed of the alternation of strychnine effects and of
effects normal in character and amplitude (Fig. 64, D). In the course
of prolonged stimulation it can happen that one shock of stimulation
causes a strychnine effect and two or three shocks cause normal effects,
then again one shock causes a strychnine effect, etc. (Fig. 64, A). It
is interesting that when at local strychnine poisoning of the sensory-
motor region of the cortex stimulation of the sciatic nerve causes
alternating normal and strychnine effects, then too direct electrical
stimulation of the brain of the same frequency produces in the poisoned
part alternating intense and weak negative potentials (Fig. 64, C).
At a stimulation frequency of the sciatic nerve of 50 per second the
first shock of stimulation causes a strychnine potential, but all
subsequent ones usually cause fluctuations of biopotential of smaller
amplitude, following the rhythm of the stimulation (Fig. 64, B).

(Legend to Fig. 64, textp. 154: Effect of strychnine on primary
responses arising at stimulation of the sciatic nerve. Cat No. 32.

5 minutes after local strychnine poisoning (1% solution) of a discharge
point of the gyr. sigmoideus post. A - the sciatic nerve of the opposite
side is stimulated; intensity of stimulation 2 volts, frequency 10
per second. B - 50 per second. C - 15 minutes after poisoning; effect
of electrical stimulation of the surface of the same convolution at a
distance of 8 mm. from the point of discharge; stimulation intensity
30 volts, frequency 10 per second. D - another preparation (cat No. 13);
point of cortex (gyrus sigmoideus post.) under discharge electrode was
locally poisoned by strychnine (0.1% solution); effect of stimulation
of the sciatic nerve several minutes after poisoning; stimulation
frequency 10 per second.)

(Legend to Fig. 65, textpage 155: Strychnine responses at diverse
frequency of stimulation of the sciatic nerve. Cat No. 43. The potentials
are discharged from a point of the gyrus sigmoideus post. poisoned by
strychnine (1% solution). The sciatic nerve of the opposite side is
stimulated. A - frequency of stimulation about 2 per second. B - about
12 per second, greater rate of exposure. C - also at a lower rate of
exposure.)

When the functional state of the cortex is good, intensified
strychnine effects can arise at a nerve-stimulation rhythm of up to
15 per second (Fig. 65). In Fig. 65 attention is turned to the fact
that at infrequent stimulations ordinary effects arise (+-+); at a

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frequency of 10-15 per second the character of the bioelectrical

potentials arising changed: additional negative potentials appeared

(compare recordings A and B of Fig. 65).

Electrical potentials discharged from various layers of the

cortex at stimulation of the sciatic nerve. The following series of

experiments was set up with sinking of the microelectrode deep into

the cortical substance. Fig. 66 illustrates such an experiment. At

first the potentials that arose at single stimulations of the sciatic

nerve were registered when the position of the microelectrode was on

the surface of the cortex. As seen from recording A, with a latent

period of 7 milliseconds a positive fluctuation of potential arose,

after which a negative followed, considerably less in amplitude. At

sinking the microelectrode to a depth of 0.6-0.7 mm. the character

of the effect was characteristically altered (recording B): 7 milliseconds

after the artefact of stimulation a negative fluctuation of potential

arose of the same length as the positive fluctuation of the surface

effects; then a positive phase followed that corresponded to the negative

phase of the surface effects.

Since the negativity of the potentials registered testifies to

the excitation of elements found in contact with the discharge electrode

or in direct proximity with it, then on the basis of the experiments

just considered with discharge of potentials from the surface and from

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the depths of the cortex it is necessary to conclude that a positive

potential, registered from the surface of the cortex, expresses by

itself excitation of the elements of the deep layers of the cortex.

The relationships of the deep effects to tetanization are the

same as those of the surface effects.

Experiments with simultaneous registration of potentials of

various layers of cortex in more conclusive form demonstrate the

correctness of the hypothesis which was made on the basis of experiments

in which the potentials of the various layers were studied with one

microelectrode, which later on was sunk deeply into the cortex.

On the basis of experiments, the recordings of which are

presented in Fig. 67, in which the first microelectrode was found at

the surface of the cortex and the second microelectrode was found in

the cortex at a depth of 0.6 mm., at a distance of 1 mm. horizontal

from the first, it is possible to make the following conclusions:

1) The so-called "spontaneous" electrical activity has identical

character at its discharge from the surface of the cortex and from the

depths of the cortex. Potential fluctuations are entirely synchronous

and directed identically. However, their intensity at discharge from

the surface is somewhat greater (Fig. 67, B). It is impossible to

ascribe these differences in intensity only to difference in discharge

conditions, which is seen from the following experiments with discharge

of electrical activity of different layers of cortex when both

electrodes are sunk deeply into the cortical matter.

2) At stimulation of a nerve many-phase potentials arise which

are an inverted reflection of one another (Fig. 67, A). It is entirely

likely that if the second electrode was found precisely under the first,

then it would register effects which would be indeed accurate, but an

inverted reflection of the surface ones.

3) At tetanic stimulations with a frequency of 10 per second

these comparatively complex effects begin to alternate with simple

ones that are only the first phases of the corresponding (surface

and depth) effects: from the surface a positive fluctuation is

discharged and from the depths a negative fluctuation corresponding

to it (Fig. 67, C).

4) On a background of slow negative fluctuations discharged

from the depths of the cortex quick ones are arranged that reach the

greatest amplitude and frequency at the height of the slow negative

potential (Fig. 67, A_1).

Thus, when one electrode is found on the surface of the cortex

and the second at a depth of 0.6-0.7 mm., then with the arrival at

a given point of the cortex of a discharge of afferent impulses the

surface electrode discharges a slow positive potential, but the

electrode in the depths discharges a slow negative potential, complicated

with quick impulses.

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(Legend to Fig. 66, textp. 156: Bioelectrical potentials discharged from different layers in the surface of the skin analysor at stimulation of the sciatic nerve. Cat No. 13. A - microelectrode on surface of gyrus sigmoideus post. in the region of the projection of the skin of the hind paw; effect of one shock of stimulation applied to the sciatic nerve of the opposite side. B - the micro- electrode was sunk to a depth of 0.7 mm.; effect of the same stimulation (Roitbak, 1953b).)

(Fig. 67, textp. 157: Bioelectrical potentials discharged simultaneously from different layers of the cortex in the territory of the skin analysor at stimulation of the sciatic nerve. Cat No. 13 twenty-five minutes after the (0.1%) strychnine was removed from the portion of the cortex being investigated; 1st microelectrode is found on the surface of the cortex, 2d microelectrode at a depth of 0.6-0.7 mm., at a distance 1 mm. horizontally from the first electrode. The potentials are discharged simultaneously from the surface (lower curves) and from a depth of 0.7 mm. (upper curves). The sciatic nerve of the opposite side is stimulated. A - frequency of stimulation about 5 per second; A_1 - effect of first shock of stimulation in enlarged form; B - "spontaneous" activity; C - frequency of stimulation about 14 per second.)

In the experiment the recordings of which are presented in

Fig. 68, A, one electrode was sunk to a depth of 0.2-0.3 mm., the

second to a depth of 0.9-1.0 mm. At these levels other phenomena

were observed: from the upper electrode in response to stimulation

of the nerve no definite bioelectrical potential was discharged;

from a depth of 1 mm. a negative potential was registered. As for

the "spontaneous" fluctuations of potential, the upper electrode

discharged potentials of greater amplitude than did the electrode

sunk 1 mm. deep into the cortex. In the experiment the recordings

of which are presented in Fig. 68 the electrodes were sunk in 0.5 mm.

further. Hence, the upper electrode was found at a depth of 0.7-

0.8 mm. and the lower at a depth of 1.4-1.5 mm. Now at stimulation

of the nerve the upper electrode discharged a considerable negative

potential and the lower electrode discharged a negative potential

also, but attenuated in comparison with the potential which was

discharged when it was higher (at a depth of 0.9-1.0 mm.). The

upper electrode now also discharged "spontaneous" potentials of

greater amplitude than the lower (Fig. 68, C).

On the basis of the experiments just considered with registration

of potentials of various layers of the cortex it is possible to make

the following conclusions:

1) At the arrival in the cortex of a discharge of afferent

impulses certain neuronic elements arranged at a depth of 0.6 mm. and

lower from the surface of the cortex generate a slow negative potential;
the main source of negativity is found at a level 0.6-0.7 mm. from the
surface; the "inversion" of potential occurs at a level 0.3-0.4 mm.
from the surface, from which this potential is already discharged in
positive form.

2) A negative slow potential arising after an initial positive
one (discharge from the surface of the cortex) is linked chiefly with
the activation of neuronic elements of the surface layers. At sinking
of the electrode to (a depth of) 0.2-0.3 mm., it is not registered
(Fig. 68, A). When the electrode is sunk into the middle layers,
then it is registered in the form of a positive potential that sets in
after the initial negative one (Fig. 68, B), i.e. the "inversion" of
this potential occurs somewhere in the surface of the cortex, evidently
also at the level of 0.3-0.4 mm.

In the experiments considered above with simultaneous discharge
of potentials from different layers of the cortex the discharge was
unipolar: potentials of electrodes in different layers of the cortex
were not related to one another and to the "indifferent" electrode
(Fig. 69). In a technical sense there are no indifferent points in
the preparation (Kogan, 1949), however, as numerous experimental data
show, a needle fastened to bone over a frontal air-sinus of narcotized
animal is practically an indifferent electrode.

(Legend to Fig. 68, textp. 158: Bioelectrical potentials discharged simultaneously from different layers of the cortex in the region of the skin analysor at stimulation of the sciatic nerve. Cat No. 13, another portion of the gyrus sigmoideus post., prior to experiments with local strychnine poisoning. A - biopotentials are discharged by two microelectrodes simultaneously from different layers of the cortex; the first electrode was sunk to a depth of 0.2-0.3 mm. (lower curve), the second to 0.9-1.0 mm. (upper curve); effect of single stimulation of the sciatic nerve of the opposite side. B - electrodes are sunk in a further 0.5 mm.; now the first electrode (lower curve) is sunk to a depth of 0.7-0.8 mm., the second to 1.4-1.5 mm.; the effect of the same stimulation. C - "spontaneous" activity (Roitbak, 1953b).)

(Legend to Fig. 69, textp. 159: Schemes of unipolar and bipolar discharge of biopotentials of the cortex. A - scheme of discharge from different layers of the cortex used in the present investigation during a study of bioelectrical potentials arising during peripheral stimulations, during stimulations of the surface of the cortex, as well as during a study of "spontaneous" electrical activity. B - scheme of discharge in analogous experiments of Bishop and coworkers. E_1 and E_2 - discharge electrodes; i - the indifferent electrode.)

Bipolar method of discharge limits the possibilities of analysis

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and can lead to incorrect conclusions. In the case in question those

methods of discharge are intended during which both electrodes are in

a part of the cortex excited by one or another stimulation, i.e. they

are methods of discharge with the interpolar distance small, measured

in mm., for instance when the pair of discharge electrodes is placed

on the surface of the cortex and the potentials arising at stimulation

of the surface of the cortex (Adrian, 1936) are registered bipolarwise,

or when the first discharge electrode is found in one (layer) and the

second in another layer of the cortex in any projection territory

whatsoever, and the potentials that form during corresponding peripheral

stimulation are registered bipolarwise (Bishop, 1936-1953). A series

of difficulties of interpretation of the recordings obtained arises

with such a method of discharge.

1) For instance, electrode 1 is found in layer IV and electrode

2 in layer II; when a discharge of afferent impulses comes to a given

point of the cortex, electrode 1 becomes negative in respect to

electrode 2. However, what is the condition under electrode 2? Well,

then, electrode 2 will be positive in respect to electrode 1 even in

this case if activation of neuronic elements again occurs under them

and even if less intense activation occurs here than under electrode 1.

Hence, on the basis of the fact that electrode 2 is positive in

respect to electrode 1, it is still impossible to clarify what the

state is of the point of the cortex under electrode 2. Or, for

example, two discharge electrodes are placed on the surface of

the cortex, electrode 1 closer to the portion stimulated, electrode

2 farther from it. At stimulation of the cortex, electrode 1

becomes negative in respect to electrode 2. However, this is

perhaps governed either by the fact that electrode 2 stands already

at such a point of the cortex where no negative potential arises

(i.e. excitation of the top dendrites) during a given stimulation of

the cortex, or by the fact that under electrode 2 a negative

potential of less amplitude arises than under electrode 1. In

both cases electrode 2 will be positive in respect to electrode 1.

Thus, with the method of discharge being considered, during

which potentials of the active points are related to one another,

it is possible to ascertain only the gradient of the potential.

2) If at both points of the cortex (under electrode 1 and

under electrode 2) biopotentials arise simultaneously, identical

in intensity, configuration, and sign, then, at relating these

isopotential points to one another, it is generally not possible

to discover the biopotential. It is known, for example, that

when the 1st electrode is on the surface of the skin-muscle projection

region and the 2d at any part of the cortex, however within the limits

of the projection region in question, then at stimulation of the

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corresponding sensory nerve "primary responses" of a certain amplitude
are registered; their amplitude is not reduced at shifting electrode
2 over the cortex as long as it does not fall into the projection
region in question; when it is established here around electrode 1,
then the amplitude of the potentials being registered is sharply
reduced (Gardner and Haddad, 1953). Or, for example, when both
electrodes are found at a number of horizontal points of the auditory
projection region, then in response to a sound shock no fluctuation
of the biopotential is observed (Pribram and coworkers, 1954).

3) If at both points of the cortex (under electrode 1 and
under electrode 2) biopotentials arise, out of phase for instance,
so that the potential under electrode 2 forms while the fluctuation
of potential under electrode 1 has not yet terminated, then an
abnormal curve is obtained owing to the algebraic summation of
these potentials. For instance, in the experiments of Marshall and
coworkers (1941) a sharp distortion of form of the response bio-
potentials was observed when the latter were discharged bipolarwise,
but both electrodes were in the excited portion of the cutaneous
projection region. This distortion of form of the biopotentials
evidently is linked with the fact that at different points of the
portion activated direct bioelectrical effects set in with a different
latent period.

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The "monopolar" method of discharge is devoid of these
deficiencies. With this method one electrode (the "indifferent")
is established in bone or at such a point of the cortex where
excitation of neurons does not arise in response to the stimulation
applied, the direct effect of which they wish to study, and the
potentials of the other ("active") discharge electrodes refer to
it.

Bioelectrical potentials arising in auditory projection
region of the cortex at sound stimulations. In these experiments,
certain results of which will be stated below, potentials were
discharged from the dura mater through electrodes attached to the
bone of the skull. In some cases a method was used which Tskipuridze
(1950a) proposed: silver electrodes, covered with cambric were,
established through small openings in the skull, on the dura mater and
clamped in this position. In other cases a "button" of plexiglass
with electrodes built into it was inserted into a trepanation opening.
The electrodes were established so that one of them, the "active",
was over the auditory projection region of the cortex, the other was
outside it, for instance over the gyrus suprasylvius med. Such a
method of discharge gives an opportunity to register unaltered primary
responses of the projection region in question. If both discharge
electrodes are in the projection region, then, first, a certain
summary effect is registered (see above) and, secondly, it is impossible
to foresee the sign of the potential being discharged. Such a principle

of discharge of biopotentials, during which both electrodes discharge
the electrical activity of the cerebral cortex but only one of them
is "active" during the investigation, was used in EEG investigations
on people during a study of the alpha-rhythm (Roitbak and Savaneli,
1952, 1953) and for study of direct bioelectrical reactions to skin
stimulations (Dawson, 1947, 1954a).

Sound shocks served for stimulation. Their intensity and
frequency can be changed within wide limits.

(Legend to Fig. 70, textpage 161: Effect of sound stimulations
during deep nembutal narcosis. Cat IV, June 3, 1952. The electrodes
were established on the dura mater through fine openings in the skull.
The position of the electrodes is indicated in the scheme. Biopotentials
are discharged simultaneously from electrodes E_1 and E_2 (lower curves)
and from electrodes E_3 and E_4 (upper curves). 2 hours after operation.
A - "spontaneous" electrical activity. B - effect of sound shocks of
a frequency of 1.5 per second. C, C_1, and C_2 - effects of sound shocks
at greater rapidity of exposure.)

(Legend to Fig. 71, textp. 162: Effect of sound stimulations
during light narcosis. The same cat on the day following the operation.
Cat drunk, sound stimulations not perceived and cause no outer reactions;
in response to tweaking a paw and blowing breath into an ear, active
reflexes. A - effect of sound shocks with a frequency of 1.5 per second.

B and B₁ - effects of sound of clapping the hands. C - recording

of June 5, 1952; cat freed of narcosis; "spontaneous" activity in

wide-awake state of animal.)

At relatively deep nembutal narcosis when a certain picture

of "spontaneous" electrical activity was observed that is characterized

by periodically arising groups of slow fluctuations at a rhythm of

about 10 per second (Fig. 70, A and B), in response to sound shock a

typical primary response was registered: 10 milliseconds after the

moment of stimulation a positive potential arose, after which a negative

fluctuation of small amplitude sometimes set in (Fig. 70, B and C).

In the motor territory of the cortex, in response to sound stimulations,

direct bioelectrical effects (primary responses) did not arise.

When the narcosis attenuated, the sound shocks began to produce

more complex effects: after a positive potential a prolonged intense

negative potential set in (Fig. 71, A). Even more complex effects

arose at the sound of the clapping of hands (Fig. 71, B).

At a sound-shock frequency of 5-10 per second the amplitude of

the response potentials was reduced. With deep narcosis quick abatement

of them was observed in the course of stimulation. At a shock frequency

of 50 per second the switching on of the stimulation provoked an effect

similar to the effect at one sound shock, and then the stimulation

remained, as it were, ineffective. It should be noted that in the

cutaneous projection region of the cortex in narcotized animals it was

possible to observe a rhythmical bioelectrical effect at this stimulation
frequency of the sciatic nerve.

The primary responses to sound were registered only in the gyr.
ectosylvius ant. and gyr. ectosylvius med. and in the upper part of
the gyr. sylvius; they were not observed in the gyr. ectosylvius post.
and in the gyr. suprasylvius.

Thus, in response to sound stimulations in narcotized animal
direct bioelectrical effects arise in a limited territory of the cortex.
With deep narcosis the reaction is limited by the arising of a positive
potential about 10 millisec. in length. With light narcosis after the
positive potential a negative potential arises. Thus, primary responses
of the sound projection region of the cortex are extraordinarily
similar in character to the primary responses of the skin projection
region of the cortex that arise with stimulations of the skin or of
the sciatic nerve.

Histological information on cortical layer IV. Afferent fibers.
In layer IV mainly afferent fibers of the cortex terminate which
belong to the so-called specific afferents, for instance fibers that
proceed from the corpus geniculatum laterale to the optic projection
region of the cortex, from the corpus geniculatum mediale to the
auditory projection region of the cortex, etc., i.e. these fibers are
connected with the sending of impulses at excitation of the analysors -

optic, auditory, skin, etc. Each afferent fiber, not dividing and

not losing its myelin covering, ascends through layers VI and V.

Upon reaching layer IV it divides dichotomously, then it loses its

medullated membrane and forms very fine branching that terminates

synaptically on neuronic elements in layer IV, and only certain

ramifications enter into layer III (Lorente de No, 1943; O'Leary, 1941).

However, although the main mass of synaptic endings of the specific

afferent nerves is found in layer IV, it is thought that other layers

too have connection with the specific afferents. It is difficult to

recognize that layers I and II/connection with the specific afferents
have

(Lorente de No, 1943).

The neurons of layer IV. a) Pyramidal neurons of layer IV.

They have a delicate top dendrite that enters into layer I; horizontal

dendritic branches depart from it in layer IV (Lorente de No, 1943).

Axons of the pyramidal neurons of layer IV are mainly association

fibers. They give off recurrent collaterals that reach layer I and,

descending, they give off collaterals into layers V and VI. b) Neurons

with short axons. Some of them have ascending, others have descending

axon. There are also cells with a horizontal axon (O'Leary, 1941).

Different types of cells of layer IV, the axons of which ascend into

layer I, were described above during consideration of the origin of

the fibers of layer I (textpage 44). According to Bliumenau, layer IV,

of all the layers, contains the greatest number of neurons with

ascending axon.

Neurons with short axon are distinguished not only through the place of termination and the character of the branching of their axons, but also by their dendrites, and this circumstance permits eliminating many types of neurons with short axon. According to Lorente de No and O'Leary, in layer IV there are at least 8 types of neurons with short axon. In three of them the dendrites branch into layer IV, and from the five other ones (they all send an axon into layer I) long dendritic branches ascend into layer I. Thus, besides the pyramidal neurons and branches, many cells with short axon of layer IV have dendrites ascending into layer I.

It should be noted that layer IV does not receive collaterals from the pyramidal neurons of the layers lying above.

Very fine ramifications of specific afferent fibers terminate in layer IV both for the pyramidal cells and for cells with a short axon (Poliakov, 1953). Afferent fibers in layer IV divide repeatedly, and numerous branches terminating with synaptic tufts have the form of a basket or cyst and are located on the bodies of the neurons (Chang, 1952; Poliakov, 1953). (It is interesting that the afferent fibers form such type of ending even for neurons of all intermediate stations, for example in the bulbar sensory nuclei of the auditory nerve, in the corpus geniculatum mediale, in the corpus geniculatum laterale, etc. (Chang, 1952). Apparently linked with this is the fact that even

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slight peripheral stimulations, for instance stimulation of several

skin receptors, can lead to a spread of excitation to the cerebral

cortex.)

(Legend to Fig. 72, textp. 164: Schematic depiction of certain

neurons of cortical layer IV and their connections. L, 2, and 3 -

main types of neurons of layer IV. 1 - pyramidal neuron of layer IV

(association pyramid), a - its axon proceeding into the white matter;

k - return collateral proceeding into layer I. 2 - neuron with short

axon ascending into layer I. 3 - neuron with short descending axon.

4 and 5 - pyramidal neurons of layer V (efferent, projection pyramids).

AF - afferent fibers. Scheme composed on the basis of certain

histological data.)

In Fig. 72 the main types of neurons of layer IV are shown.

On the basis of the histological data presented it is possible

to make the following conclusions.

1. Direct activation of the neuronic elements mainly of layer

IV should occur at the arrival of the afferent impulses in the cortex.

Both pyramidal neurons and neurons with short axon should be activated

thereby.

2. Elements of layers I and II are not activated by afferent

impulses. Because of this, excitation of the neuronic elements of

these layers after the arrival of the afferent impulses in the cortex

can proceed only under the influence of impulses from the cortical

neurons that have been excited under the influence of the afferent

impulses, i.e. chiefly of the neurons of layer IV.

3. With the coming of the afferent impulses to the neurons

of layer IV excitation should arise in the bodies of the corresponding

neurons, and, in view of the density of the arrangement of synaptic

endings, a discharge of impulses of excitation in these neurons should

arise with great facility.

4. At excitation of the neurons of layer IV through the system

of their ascending axons and collaterals activation should occur of

the elements of layer I of the cortex. This conclusion agrees with

the general conclusion of Lorente de No (1938, cited by Zurabashvili,

1941), according to which, owing to the presence of cells with ascending

axons, at stimulation of the deep layers discharges of impulses should

be sent almost instantly to the upper layers.

5. Excitation of the pyramidal neurons of layer IV should lead

to spread of excitation through the cortex for great distances by

means of association fibers that pass into the white matter and terminate

in various regions of the cortex in layers II, III, and VI.

The origin of the initial positive slow potential of the primary

bioelectrical reaction of the cortex. Quick potentials, as a rule,

precede the positive potential registered in the optic region of the

cortex at stimulation of the optic nerve or retina. (At stimulation

of the optic nerve 3 quick fluctuations arise: the 1st, with a length

of 0.7 millisecond, is registered 1.6 milliseconds after the moment of

stimulation; the 2d and 3d are superimposed on a slow positive potential

which develops after the first quick impulse (Marshall, Talbot, and

Ades, 1943). According to Chang, these three quick fluctuations express

excitation of three groups of afferent fibers of different diameter

(Chang and Kaada, 1950; Chang, 1952a). Chang proposed a hypothesis

according to which these three kinds of fibers carry impulses of

excitation from three types of color-receiving receptors of the retina.

However, it is still impossible to consider the occurrence of the three

quick fluctuations as clarified (Cragg, 1954).) A negative potential

several milliseconds in length can precede the positive potential

registered in the auditory region of the cortex during sound stimulations

(Mickle and Ades, 1953). This initial negative potential apparently

expresses arrival in the cortex of afferent impulses (Bremer and coworkers,

1954; Narikashvili, 1955), which were recorded by Tunturi (1949) in the

auditory region of the cortex in dog. A group of "peaks", i.e of

quick fluctuations of axonic origin (Roitbak, 1953b) or an initial negative

potential (Scherrer and Oeconomos, 1954), precedes the positive potential

registered in the skin region of the cortex. At stimulation of other

receptors and of the sensory nerves afferent impulses failed to be

registered before the slow positive potential. Afferent impulses
which come to a corresponding area of the cortex are registered
from the surface of the cortex, as already said, in far from all
cases, which is probably stipulated by poor conditions of discharge
of quick fluctuations from the cortical surface.

Adrian (1941) with the help of a needle electrode sunk deep
into the cortex in the region of the cutaneous projection recorded
"impulsatinn" in the separate afferent fibers that arose in response
to skin stimulation. At tactile stimulations short discharges
arose. At stimulation of the receptors the frequency pressure of
"impulsation" reached 100 per second and a discharge could last up
to 2 minutes. Sometimes an aftereffect was observed in the form of
discharges of impulses at a rhythm of 10-20 per second. As was
explained, this aftereffect is of thalamic origin, and the impulses
issue from those cells of the thalamus which were excited by peripheral
stimulation. In the recording of Fig. 57, A, it is seen that, after
the initial group of afferent impulses, a slow positive potential
arises; however, on the background of it and for a long time at its
completion "impulsation" continued at a rhythm of about 50 per second.
It can be considered as an aftereffect from the thalamus.

As already said, the slow positive potential arises without
pause after a shock of excitation of the afferent fibers.

Consequently, the latent period of the primary responses is
determined by the time necessary for the afferent impulses to reach
the cortex. At stimulation of sensory nerves it equaled 2.3-12
milliseconds. At stimulation of the nerves of a front extremity
the latent period was shorter than at stimulation of nerves of a
hind extremity. At stimulation of the chorda tympani, a nerve containing
fine fibers, it is greater than at stimulation of the n. lingualis that
contains thick fibers too (10-12 milliseconds and 6-8 milliseconds,
respectively). At adequate stimulations of the receptors the latent
period is very lengthened, since the time needed for excitation of
the receptors is added. For example, the latent period of the primary
bioelectrical reaction of the optic area of the cortex in response
to exposure of the eye to light equals 17-25 milliseconds, and in
response to electrical stimulation of the optic nerve 2.3 milliseconds
(Marshall and coworkers, 1943).

The majority of investigators think that the initial positive
potential is a cortical phenomenon, i.e. that it is connected with the
activity of the cortical neurons (Gershuni, Bremer, Eccles, Artem'ev,
Chang, Beritov, et al.). Still there is a hypothesis that it is
connected with excitation of the afferent fibers of the cortex (Adrian,
1941; Monnier, 1952). The greater amplitude and length of the initial
positive potential is explained by the fact that it reflects not so

much the reaction currents of the afferent fibers as the subsequent
potentials of these fibers. Finally, the hypothesis has been expressed
that this potential reflects the activity not of the cortical but of
the thalamic elements and that it is the result of electrotonic spread
of potentials from the corresponding nucleus of the thalamus along
the thalamo-cortical fibers (Adrian, 1941). However, in the first
place it is difficult to explain from this point of view the fact
of the intensification of the positive potential at strychninization
of the cortex and, in the second place, the positive potential
(expression of the depolarization of the thalamic cells) must some-
what precede the afferent impulses (i.e. the discharge of the thalamic
cells), because from this point of view it is the cause of their arising.

The following facts speak in favor of this, that the initial
positive potential is an expression of the activity of the cortical
neurons.

1) It is intensified at local application of a number of substances
to the cortex: a) Strychnine. At local strychnine poisoning the
amplitude of the positive potential of the primary responses provoked
in the skin area of the cortex at stimulation of the sciatic nerve is
increased (Fig. 63). This was likewise ascertained in respect to the
primary responses of the optic region (Chang and Kaada, 1950) and of the
auditory area (Bremer, 1943, 1952). b) Veratrine (Goldring and O'Leary,

1954). c) Atropine (Chatfield and Purpura, 1954). d) A 1% solution of novocain (Goldring and O'Leary, 1954).

2) It abates at local application of a number of substances to the cortex: a) Novocain (3% solution). At local poisoning with novocain the positive potential can disappear; the quick biocurrents preceding it cannot thereby be altered (Chang and Kaada, 1950). b) Acetylcholine (Artem'ev, 1951; Chatfield and Purpura, 1954).

3) If the discharge electrode shows pressure on the cortex, then this leads in time to complete elimination of the positive potential; the quick biocurrents of the afferent endings are not thereby changed (Chang and Kaada, 1950).

4) When the wave of slow-spreading depression of cortical electrical activity reaches a given projection region of the cortex, the primary bioelectrical effects produced by corresponding peripheral stimulation are sharply attenuated (Leao, 1944; Marshall, 1950); in particular, the initial positive potential attenuates; the quick biocurrents preceding it are unaltered.

All these data indicate that the positive slow potential is stipulated by excitation of the cortical neurons. (Abatement or disappearance of the positive potential during anoxia (Chang and Kaada, 1950), during anemia, and under the effect of curare (Ostow and Garcia, 1949) can be stipulated not only by a worsening of the functional state of the cortical neurons but by abatement or cessation

of impulses from subcortical cells, which also undergo injurious

influences when there are these two effects on the organism, so that

these facts cannot serve as direct proof that the positive potential

reflects the activity of the cortical neurons.)

When a positive potential arises in pure form, i.e. when no

slow negative potential follows after it, then it lasts about 10

milliseconds. The length of the reaction being considered agrees

well with the position that it is the result of local potentials.

As we have seen, a magnitude of the order of 10 milliseconds

characterizes the length of local potentials in the neuronic elements

of the various parts of the central nervous system.

At sinking the discharge electrode deep into the cortex the

maximal negative potential is registered at a depth of about 0.6 mm.,

i.e. at the level of layer IV. This potential is localized, since

at the level of about 0.4 mm. an "inversion" of the potential occurs:

here no definite potential is discharged, but from the surface (from

layer I) a positive potential is discharged.

The facts which Amassian (1953) obtained by using glass electrodes

1.2-12 microns in diameter fully correspond to facts which were obtained

in the experiments described above (with electrodes 60-80 microns in

diameter). According to his data, in response to stimulation of a nerve

from the surface of the cortex and from a depth of up to 0.2 mm. (from

layer II) a positive potential is registered, the amplitude of which

at sinking the electrode more deeply progressively diminishes (Fig.

73 - 1, 2, and 3). From a depth of 0.6-0.95 mm. (from layer IV) a

negative potential is registered, complicated by high-voltage quick

potentials (Fig. 73 - 4, 5, and 6). The negative potential has

greater amplitude in layer IV than in layer V (at a depth of 1.2 mm.).

(According to the data of Cragg (1954), "inversion" of potential is

observed at a depth of 1.25 mm., but maximal negative potential

is registered from a depth of 2.25 mm. (i.e. under the cortex, inas-

much as the thickness of the cortex in the region of the gyrus

sigmoideus post. equals 1.8 mm. according to the data of Li and

Jasper, 1953).)

(Legend to Fig. 73, textp. 168: Bioelectrical potentials

registered by means of a microelectrode 1.2 microns in diameter

from different layers of the cortex in the region of the skin

analysor at stimulation of the ulnar nerve. 1 - electrode on the

surface of the cortex. 2 - electrode sunk to a depth of 120

microns (layer I). 3 - electrode sunk to 200 microns (layer II).

4 - to 600 microns; 5 - to 700 microns (layer IV); 6 - to 950

microns (boundary of layers IV and V); 7 - electrode in white

matter, at a depth of 2650 microns (according to Amassian, 1953).)

Experiments with the deep sinking of the discharge needle

electrode into the optic cortex (Marshall and coworkers, 1943;

Cragg, 1954) and into the auditory cortex (Artem'ev, 1951; Narikashvili, 1955) gave in general the same results: change of sign of potential, maximal negativity in the middle layers of the cortex, etc.

Bishop and coworkers at investigation of the optic area of the cortex used, as said, another scheme of deflection of biopotentials from various layers of the cortex; but they also came to the conclusion that the positive slow potential arising at stimulation of the optic nerve indicates the negativity of the middle layers, i.e. of the layers where the afferent fibers end (Bartley, O'Leary, and Bishop, 1937; O'Leary and Bishop, 1938).

Thus, under the influence of afferent impulses arriving in the cortex, local excitation and a local potential corresponding to it arise in the bodies of neurons of layers IV and III.

Earlier, on the basis of experiments with stimulation of the surface of the cortex and with registration of potentials from different layers of the cortex and from different points of its surface, a conclusion was made on the extraordinary local quality of the biopotentials that arise in the top dendrites. The same conclusion can be made with regard to the local potentials that arise at the arrival of afferent impulses in the cortex: local excitation is limited by the bodies of the neurons of layer IV (inasmuch as the negative potential is not registered from the top layers) and a local potential is not spread upward from the cellular bodies through the dendrites. On the other

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hand, local potentials arising in the bodies of the neurons of layer

IV are not spread horizontally to neuronic elements lying in a row.

In the cortex at stimulation of a small group of afferent fibers a

focus of a territory of approximately 0.5 mm^2 arises, from which

response potentials maximal in amplitude are registered. At drawing

the discharge electrode back 0.5-1 mm. the amplitude of the potentials

is reduced, but the effects deflected at these adjacent points are

not a consequence of physical or electrotonic spread of potentials

from a focus of maximal activity, since the response potentials here

set in with a different latent period (Marshall and coworkers, 1941;

Adey, Carter, and Porter, 1954). Thus, a local potential is registered

namely at that point of the cortex where a discharge of afferent

impulses comes, i.e. it spreads neither in a purely physical, nor

vertical, nor horizontal direction. The territory of its deflection

agrees with the territory of its arising. (As indicated, at sinking

a microelectrode down lower than layer IV, the amplitude of the

negative potential progressively diminishes. However, it continues

to register even at a depth of 4 mm., i.e. from the white matter.

This was observed in my experiments. I was able to come to this

conclusion on the basis of the recordings of Marshall (1941), Amassian

(1953), Ades and Brookhart (1950), Narikashvili (1955), Verzeano,

Lindsley, and Magoun (1953), and Curtis (1940). Perhaps the slow

negative potential discharged from the subcortical fibers, agreeing

in time with the negative potential in the cortex, is the result of

electrotonic reaction of the fibers in question, stipulated by a

local potential of the cortical neurons, on which the fibers in

question terminate or from which they have their start (see Chapter I).

This fact has not attracted attention and requires special experimental

analysis.) However, from the surface of the cortex a positive potential

is registered at this time.

At the arising of a local potential in the body of a neuron of

layer IV a difference of potentials arises between the body of the

neuron and its dendrites. It is necessary to assume that the positive

potential discharged from the surface of the cortex is the result of

the positive polarization of the dendrites ascending into layer I

from the cells of layer IV (dendrites of the association pyramids and

of the fusiform cells, as well as the dendritic branches of a number

of neurons with short axon). Thus, by stipulating the positive

polarization of the ascending dendrites the local potential of the

cellular bodies of layer IV receive their electrical expression at

deflection from the surface of the cortex. (There are facts which

indicate that at the arising of local excitation (local potential)

in the bodies of the cells of layer IV an electrotonic reaction occurs

of the axons of these cells that proceed into the white matter (see

above). On the other hand, a positive polarization of the dendrites
of these cells occurs thereby. Thus, the impression is created that
the local potential in the body or the dendrites, causes perielectrotonic
reaction of the unexcited part of the cellular soma; the local potential
of the body of the cell provokes an electrotonic reaction of its axon.
However, the unmedullated fibers, particularly axons of cells with
short axon, ought not develop an appreciable electrotonic reaction:
it is well known that in unmedullated fibers the electronus spreads
for a lesser distance than the local potential (Hodgkin, 1938).)
Thus, the characteristic positive potential registered from the surface
of the projection territories of the cortex in response to peripheral
stimulation is the expression of regional excitation of the cellular
bodies of layer IV, which arises under the effect of the biocurrent
of the synaptic endings of the afferent fibers.

We have seen that the dendritic potential, i.e. the local
potential, which arises in the top dendrites at stimulation of the
cortical surface is somewhat intensified under the action of 0.1%
strychnine. The positive potential at local strychnine poisoning
of the cortex is also somewhat intensified, it being intensified
more during the action of a 0.1% solution than of a 1% solution
(Fig. 63). This fact can serve further as an illustration that
regional excitation can be intensified under the effect of strychnine.

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Concerning alteration of the positive potential at intensification
of peripheral stimulation. At intensification of peripheral stimulation
the amplitude of the positive potential is usually increased. (For
simplicity of analysis in this paragraph only such cases are considered
wherein, because of one reason or another, reaction is limited to
peripheral stimulations by the arising in the projection territories
of the cortex of pure positive potentials, after which no additional
fluctuations of biopotential follow.) At intensification of peripheral
stimulation increase occurs of the number of peripheral fibers excited.
The fact that this occurs in case of direct electrical stimulation of
a sensory nerve requires no explanation. However, even at stimulation
of the skin with one short electrical stimulus the intensification of
stimulation leads to irritation of a greater number of skin receptors
(Dzidzishvili, 1948)[*]; at sound shock of various intensity a different
number of auditory receptors is excited (Gershuni, 1940a). ([*]Not to
speak of the fact that at intensification of electrical stimulation
of the skin different kinds of skin receptors begin to be irritated.)
At increase of the number of the excited peripheral fibers excitation
of a greater number of thalamic neurons occurs and, as a consequence
of this, increase of the number of neurons activated in layer IV,
because to each neuron of layer IV collaterals from many afferent
fibers come[*], and in one and the same cells of layer IV at intensification

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of the peripheral stimulation, i.e. at increase of the number of

excited afferent fibers, a more energetic regional excitation can

develop. (*This issues from the works of Marshall and coworkers

(1941), Adrian (1941), Amassian (1953), et al.)

Furthermore, at intensification of stimulation of the skin

receptors frequency increase of excitation impulses occurs (Adrian,

1932; Dzidzishvili, 1948). At increase of intensity of sound

stimulation the number of impulses increases in the separate

fibers of the auditory nerve (Galambos and Davis, 1943; Tasaki,

1954).

Frequency increase of afferent "impulsation" should lead,

on the one hand, to increase of intensity of regional excitation of

the given neurons of layer IV of the cortex. On the other hand,

this can lead, because of summation of excitation in the neurons

of the thalamus, to increase of number of the thalamic neurons

excited and to increase in the number of cortical afferents excited,

i.e. to the phenomenon already considered above.

Thus, at different intensities of peripheral stimulation

there occurs not only activation different in intensity of one and

the same cortical neurons but also activation of a different number

of neurons, since at intensification of the stimulation additional

neurons are drawn into reaction. In other words, at different

intensities of peripheral stimulation complexes of neurons different

in a quantitative respect are activated in the cortex already directly

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under the effect of the afferent impulses.

In experiments on deeply narcotized animals this is electro-
graphically expressed in the fact that at intensification of the
peripheral stimulation first an increase of the amplitude of the
positive potential occurs (which the "primary response" of a given
projection territory of the cortex amounts to in these conditions
of experiment); secondly, an increase of peripheral stimulation
leads to a certain increase of the territory from which the positive
potentials are discharged. (This issues from the data of Mickle
and Ades, 1952)

As said, at intensification of the peripheral stimulation the
amplitude of the p̃ositive potential is increased, but the limit
is reached very quickly and further intensification of stimulation
does not lead to increase of the biopotential. Furthermore, attention
is turned to the fact that both at stimulation of the fine nerves and
at stimulation of the thick nerve trunks in the cortex biopotentials
arise of approximately one order in the sense of their amplitude.
At electrical stimulation of a small portion of skin, i.e. at
stimulation of a small number of skin receptors, in the skin projection
territory potentials arise the magnitude of which frequently approximates
the magnitude of the potentials registered in this territory at energetic
electrical stimulation of the sciatic nerve (Figs. 58 and 59). This

paradoxical phenomenon can be easily explained by proceeding from the
fact that in different parts of the cutaneous projection territory of
of the cortex different parts of the skin surface are represented and
that the bioelectrical potentials are extremely local. At a given
point of the cortex a small portion of the skin is represented (for
example, of the hind paw). At stimulation of this portion at a
given point of the cortex a focus of greatest activity arises, i.e.
here biopotentials of the greatest amplitude are registered. At
maximal stimulation of the sciatic nerve the bioelectrical potential
at a given point of the cortex is not increased much, since here
mainly the effect of the excitation of fibers proceeding into the
composition of the sciatic nerve from the corresponding part of the
skin is registered. Excitation of numerous fibers of a nerve trunk
proceeding from other parts of the skin leads only to a little
intensification of the biopotential of a given point of the cortex.
Certain intensification of the biopotentials is connected with the
convergence of afferent fibers. Point A of the skin is not represented
by point a in the cortex; point a is excited not only at stimulation
of point A of the skin but also to a certain degree by stimulation of
a point of the skin lying in line (Marshall and coworkers, 1941;
Adrian, 1941). Amassian (1953), who showed that a given neuron from
the skin projection territory of the cortex can be activated at stimulation

of different nerves of a fore extremity, gave direct proof of the
convergence of afferent fibers at single neurons.

Thus, energetic stimulation of the sciatic nerve leads to
activation of a large portion of the cortex corresponding to the
projection for the skin of a hind extremity; but at each point of the
territory activated the amplitude of the response biopotentials
does not considerably exceed the amplitude of potentials arising
at stimulation of small portions of the skin. The same phenomenon
is observed also at stimulation of other analysors, and, finally,
in these cases the same explanation must be given to it.

Variability of "primary responses". As was indicated, the
primary responses change from one peripheral stimulation to another
in regard to amplitude, length, and configuration. It is well
known that nerve fibers too disclose "spontaneous" changes of
excitability, which is apparent in fluctuation of the thresholds
of their stimulation. One would think that, at adequate or electrical
stimulation of the receptors, stimulation shows on some of them a
near-threshold effect and, because of "spontaneous" changes of their
excitability, some of them would then be excited; this is not so.
As for the central nervous system, identical discharges of impulses
proceed from the periphery. However, this phenomenon also occurs
when a nerve trunk is stimulated by electrical stimuli of maximal

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intensity, i.e. the variability of the primary effects cannot possibly

be attributed to a change of the excitability of the receptors. It

is possible to assume that the alterability of direct cortical bio-

electrical reactions depends on changes of excitability of the subcortical

centers, particularly of the corresponding nuclei of the thalamus, because

of which in the cortex, in response to one and the same peripheral

stimulations, discharges of afferent impulses, different from moment

to moment, set in. However, it is possible, all the same, to conclude

that the phenomenon being considered reflects changes of the excitability

of the neuronic elements of the cortex. The following observation made

by Bishop and Clare (1953) speaks in favor of this. In the optic

territory of the cortex, in response to single stimulations of the

optic nerve following one another, primary responses arose in the form

of positive potentials, the amplitude of which changed from one stimula-

tion shock to another: sometimes negligible potentials arose, sometimes

quite considerable ones. However, the magnitude and character of the

preceding quick biocurrents (afferent impulses) remained strictly constant

at this. Thus, in response to identical discharges of afferent impulses

coming to the cortex, slow positive potentials of very different magnitude

can arise.

The variability of the primary responses is especially marked at

light narcosis. At deep narcosis, when "spontaneous" electrical activity

of the cortex is depressed, peripheral stimulations produce stereotypic

bioelectrical reaction. It has been demonstrated that variability
of primary responses is connected with "spontaneous" electrical activity
(Marshall and coworkers, 1941). Chang (1951) established that the
amplitude of the primary response of the auditory territory of the
cortex periodically increases and abates in respect to one or another
phase of "spontaneous" waves of the type of the alpha-waves; the
period of cyclic changes of the amplitude of the primary response equals
100 milliseconds, i.e. the length of the alpha-wave. The positive
potential has the greatest amplitude if it develops at the moment
when the alpha-wave reaches the summit, and it has minimal amplitude
when the alpha-wave damps. According to Chang, this is the expression
of cyclic changes of cortical excitation stipulated by the activity
of the nerve circles of the cortex-thalamus.

Up to now it has been emphasized that in the different projection
territories primary responses identical in character arise, but there
are differences among them. One of them consists of this, that in
the different projection territories of the cortex the "higher rhythm"
of the primary responses is different. As said, in one and the same
animal in the skin projection territory primary responses can arise
to a stimulation rhythm with a frequency of peripheral stimulation of
50 per second, but in the auditory projection territory the primary
responses follow a lower rhythm of stimulation. The "higher rhythm"

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of the primary responses of the auditory projection territory is
higher than the optic (Narikashvili, 1954). The phenomenon described
can depend not only on the difference of functional properties of
the neurons of the different projection territories of the cortex,
but also on the difference of the mechanism of activity of the dif-
ferent thalamic nuclei. (Recently data were obtained in favor of
the cortical origin of this phenomenon (Smirnov, 1955).) Since the
question of the transmission of excitation in the separate nuclei
of the thalamus has been insufficiently studied, then it is impossible
to give a definitive interpretation to the facts described above. A
second distinction consists of the following. In the course of
narcotization or at asphyxiation the primary responses constantly
attenuate and, finally, dwindle to nothing, but in the different
projection territories of the cortex the answering bioelectrical
reactions do not cease simultaneously. At deepening of nembutal
narcosis the amplitude of the primary responses that arise at stimulation
of the vestibular nerve quickly diminishes, and they cease to arise at
that depth of narcosis at which other forms of stimulation give primary
responses in the corresponding projection territories (Mickle and Ades,
1952). At inhalation of carbon dioxide the primary responses greatly
attenuate at optic stimulation, whereas primary responses to sound
stimulation are even intensified (Gellhorn, 1953). Narikashvili (1954)

notes greater sensitivity to narcosis of optic responses as compared
with the auditory. Easy to observe too is the phenomenon of the
greater sensitivity of primary responses of the cutaneous projection
territory of the cortex when compared with primary responses of the
auditory projection territory. Thus, the projection territories of
the cortex can be arranged in the following order on the basis of
this, how quickly they cease generating primary responses at
worsening of the functional state: vestibular, optic, auditory, and
cutaneous. Apparently this order corresponds to the order of the
falling out or infringement of the corresponding perceptions in man
at deterioration of the functional state of the brain (anoxia,
anemia, etc.) and when there are certain physiological conditions
(at the "dead center" of sportsmen). By what is this phenomenon,
the different sensitivity of the neurons of the various projection
territories of the cortex or the different sensitivity of the neurons
of the subcortical (thalamic) nuclei of the corresponding analysors,
stipulated? In the optic projection territory of the cortex under
the effect of carbon dioxide not only the primary responses quickly
attenuate, but the "spontaneous" electrical activity dwindles to
nothing, while in the auditory projection territory the primary
responses do not abate and only a certain attenuation of the "spon-
taneous" electrical activity is observed (Gellhorn, 1953). This

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fact is not fully substantiated, since the intensity of the "spontaneous" electrical activity of the cortex depends not only on the functional state of a given territory of the cortex but also on the functional state of the thalamic nucleus connected with it. However, proceeding from a well-known position that the cortical neurons are far more sensitive to different injuries than the neurons of the subcortical nuclei, it is possible to think that the different rate of disappearance of the primary responses in the different projection territories of the cortex testifies to functional differences of the neurons of layer IV of the different projection territories.

Origin of the negative potential of the primary bioelectrical reaction of the cortex. At light narcosis peripheral stimulation provokes in the corresponding projection territory of the cortex a "primary response" that consists of a positive slow potential, after which a negative slow potential follows. It is characteristic that the same effect arises in response to electrical stimulation of the middle layers of the cortex (Bishop and Clare, 1953).

A great number of facts indicate that a negative potential arising after an initial positive one is stipulated by excitation of the cortical neurons.

1) It does not arise at deep narcosis, which was noted by many authors, and when the functional state of the cortex was poor, caused for example by drop in blood pressure (Bremer and Bonnet, 1949).

2) During anoxia it disappears appreciably earlier than the

initial positive potential (Chang and Kaada, 1950). At pressing

the carotid arteries it attenuates considerably more highly than

the initial positive potential (Adrian, 1941).

3) It quickly disappears at local poisoning of the cortex by

nembutal (Marshall, Talbot, and Ades, 1943). It disappears first

and foremost at introduction of malononitrile (a compound that

gives off HCN); then the amplitude of the positive phase diminishes

only progressively (Goldring, O'Leary, and Lam, 1953).

4) It attenuates and disappears in connection with increase

of frequency of stimulation more quickly than the positive. For

instance, in narcotized animal with adequate stimulations of the

skin at a rhythm of 5-10 per second the negative potential quickly

disappears, and the stimulations begin to provoke only positive

potentials (Adrian, 1941). The same is observed in experiments with

electrical stimulation of the skin at a rhythm of 5 per second

(Scherrer and Oeconomos, 1954) and in experiments with stimulation

of a skin nerve (Chatfield and Purpura, 1954). This phenomenon is

described too in respect to primary responses to sound shocks

(Narikashvili, 1955).

5) It attenuates and disappears at prolonged peripheral

stimulations (Fig. 61).

6) It is extraordinarily intensified under the effect of
strychnine (Fig. 63). If stimulation causes only a positive potential,
then after poisoning of the cortex with strychnine it begins to cause
subsequent negative potential too (Bremer, 1949). Pyrotoxin too
influences in the same way (Marshall and coworkers, 1943).

Negative potentials discharged from the surface of the cortex,
as we have already seen, are an expression of the activation, of
the regional excitation of the neuronic elements of the surface layers
of the cortex, of elements with which the discharge electrode comes
in contact. However, excitation of the elements of the surface layers
does not exclude the possibility of simultaneous excitation of the
elements of the deep layers. For instance, when at stimulation of
the surface of the cortex after a dendritic negative potential a
supplementary negative potential arises, then not only elements of
the surface cortical layers but also elements of the deep cortical
layers are found in an active state of this: at sinking the discharge
electrode deep into the cortex change of sign of the supplementary
fluctuation does not occur and in the depths of the cortex an
additional negative potential is registered (whereas the initial
dendritic potential in the depths of the cortex changes its sign).
Or, for example, from the surface and from all layers of the cortex
synchronous "spontaneous" fluctuations of identical sign are simultaneously

discharged (Figs. 16, 67, and 68). However, in regard to the negative

potential that arises at peripheral stimulations, at laminar study

the same phenomena are observed, say, as are observed at a similar

study of the dendritic potentials arising under the effect of impulses

from cortical layer I: at sinking the electrode to a depth of 0.3-

0.4 mm. the sign of the potential being investigated is changed

(Figs. 66-68, 73). Consequently, the negative phase of the primary

response is stipulated by activation of the neuronic elements of the

surface layers of the cortex. From the middle and deep layers of

the cortex at this time a positive potential is registered. This

indicates that the negative potential is connected with activation

of the dendrites of such neurons as occupy the greater part of the

thickness of the cortex, i.e. mainly the pyramidal neurons. Experiments

with simultaneous discharge of potentials from different layers show,

furthermore, that both in case of activation of the top dendrites under

the effect of impulses from the fibers of layer I and in this case,

when they are activated by impulses from another source, local non-

spreading excitation arises in them.

The facts obtained at laminar study of the optic cortex suit

hypotheses on the localization of the potentials and on the electrical

fields arising because of excitation of the cortical cells by afferent

impulses. The negative potential, arising after the positive at

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stimulation of the optic nerve, takes possession of the upper half

of the thickness of the cortex, the maximal negative potential

being generated by elements of the upper third of the cortex (Bartley,

O'Leary, and Bishop, 1937; O'Leary and Bishop, 1938).

Likewise, the fact that it can be selectively depressed at

surface thermocoagulation of the cortex (Bremer and Bonnet, 1949;

Bremer, 1952) would indicate that the negative phase of the primary

response being considered is connected with regional excitation of

the neuronic elements of the surface layers of the cortex.

Activation of the neuronic elements of the surface layers

after arrival in the cortex of a discharge of afferent impulses

certainly is stipulated by excitation, by discharge of the neurons

of layer IV. At examination of histological data on the neuronic

composition of layer IV of the cortex, it was said that this layer

has greater possibilities for excitation of the surface layers:

in this layer there is, on the one hand, an extremely large number

of neurons with axon ascending into layer I; on the other hand,

association fibers that go out from the pyramid cells of layer IV

give off collaterals that ascend into layer I. At excitation of

the indicated neurons, the excitation impulses must be transmitted

upward and provoke in the neuronic elements of the surface layers

regional excitation, the expression of which is a negative potential

discharged from the surface of the cortex (Roitbak, 1953b). Quick

impulses precede this slow negative potential. This can be explained

by the nonsynchronous discharge of the neurons of layer IV, i.e.

the nonsynchronous arrival of impulses from the ascending axons and

collaterals to elements of the surface layers. (Compare the fact

of the absence of quick impulses before additional negative anterior-

root potentials stipulated by "impulsation" from the intermediate

neurons, as well as the fact of the absence of quick impulses before

additional negative potentials in effects caused by direct stimulation

of the cortex.)

Thus, the negative potential arises at direct stimulation of

the cortex immediately in response to stimulation, it arises during

certain conditions after the preceding, and finally it arises at

peripheral stimulations after the initial positive potential. In

all these cases it is stipulated by one cause, the arising of regional

excitation in the top dendrites of the pyramidal neurons. In the

first case the dendritic branches of the pyramids arrive at a state

of regional excitation under the influence of impulses from the

fibers of layer I; in the second case they do so under the influence

of impulses from neurons mainly of layer II (see p. 65), and finally

in the third case it is under the influence of impulses from the

neurons of layer IV. When excitation reaches the cortex along the

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callosal fibers, a negative potential must arise under the influence of
impulses from the neurons of layer III where the callosal fibers, for
the most part terminate.

Concerning the mechanism of the arising of impulses of
excitation in the neurons of layer IV. In Chapter I facts were
presented well known at the present time concerning the mechanism
of the arising of excitation impulses in the nerve cells; these
facts were obtained mainly during study of the biopotentials of
the anterior roots of the spinal cord. Motoneurons of the spinal
cord of cat at stimulations subthreshold (in the sense of the
arising of an excitation of their axons) generate local potentials
expressing their regional, nonspreading excitation; discharge of
the motoneuron into the axon occurs when, in connection with intensi-
fication or repetition of the stimulation, the local potential
reaches a certain critical size. Since usually at stimulation of
the sensory nerve only part of the motoneurons receives a sufficient
number of impulses for their discharge to occur and local potentials
to develop and die out in another part, then quick discharges on the
background of a slow potential are the typical picture at discharge
of an anterior-root potential. At deep narcosis the motoneurons are
not stimulated by impulses of excitation or during any conditions
of their stimulation.

These data can be used for clarification of the arising of
impulses of excitation in the neurons of layer IV.

Regional excitation arises under the effect of afferent
impulses in cells of layer IV. Since a slow potential arises
without pause after the afferent impulses, then evidently trans-
mission of excitation from the synaptic endings of the afferent
fibers to the bodies of the neurons is effected through currents
of excitation. If it is assumed that the neurons of layer IV are
stimulated by impulses in their own axon when their regional
excitation reaches a certain critical magnitude and that this
critical moment is the closer to the beginning of the arising of
a regional excitation the higher the excitability of the neurons
of layer IV, then it is possible to explain a whole series of
oscillographic recordings.

Analysis of the biopotentials discharged from the surface
of the cortex at the arrival of afferent impulses is quite complex
in comparison with analysis of the biopotentials of the anterior
roots of the spinal cord. At the discharging of a potential from
the axons of the motoneurons, we record bioelectrical potentials
connected with the activity namely of these neurons, the potentials
directly generated by them. In the case in question the activity
of the neurons of layer IV must be judged indirectly: as to their

regional excitation, judged on the basis of the positive potential

discharged from the surface of the cortex, i.e. of the positive

polarization of the dendrites ascending into layer I, stipulated by

the local potentials of the cellular bodies; as to discharge of

impulses into their axons, judged on the basis of the negative

potential discharged from the surface of the cortex. Moreover,

3 circumstances that complicate analysis must be taken into account.

First, far from all neurons of layer IV have dendrites ascending

into layer I. Thus, the amplitude of the positive potential

discharged from the surface does not express the actual number of

layer IV neurons activated, and it can even be assumed that at

excitation of a group of neurons of layer IV, because of the above-

mentioned circumstance a positive potential cannot be registered

from the surface of the cortex. Secondly, far from all neurons of

layer IV have axonal connection with layer I: part of the neurons

with short axon send their axons downward or horizontally; thus,

the amplitude of the negative potential discharged from the surface

of the cortex does not reflect the actual number of excited neurons

of layer IV, and theoretically it can be assumed that excitation of

a group of neurons of layer IV can occur without a negative surface

potential arising. Thirdly, regional excitation of the neurons of

layer IV and the excitation impulses spreading from these neurons

stipulate the arising of potentials of a different sign. The electrode

on the surface of the cortex registers the result of the algebraic

summation of potentials different in sign. When a positive potential

is discharged from the surface of the cortex, then this can only mean

that at the moment in question the potential of layer IV is dominant.

When a negative potential is discharged, then this can only mean that

at the moment in question a potential of the surface layers dominates.

At simultaneous discharge of potentials from layer IV and from

the surface of the cortex it is possible to state that the negative

potential of the cortical surface develops after discharge of many

neurons of layer IV. As seen in Fig. 67, the electrode in layer IV

at first registers a slow negative potential, covered with quick,

minute fluctuations. Shortly before their reaching a maximum several

powerful quick fluctuations arise. Just after this, negativity of

the cortical surface develops. Evidently at this time excitation occurs

of the main mass of neurons of layer IV that are capable of being excited

under the effect of the stimulation applied.

On the basis of known data it is necessary to think that during

heightened excitability of the neurons of layer IV first a volley of

afferent impulses causes a discharge of a larger number of neurons,

secondly a discharge with a shortened latent period arises in each

neuron, thirdly each neuron is stimulated by a greater number of

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impulses (Amassian, 1953).

It is possible to conclude that the quicker the excitation of the neurons of layer IV arises, the less will be the amplitude and length of the positive potential registered from the surface of the cortex, which can result in a short potential like a "peak". As seen in Fig. 74, the first shock of stimulation of a sciatic nerve provoked in a corresponding part of the cortex a peculiar bioelectrical reaction: the positive potential amounted to a quick fluctuation, after which a negative potential of greater amplitude arose. It is necessary to treat the metamorphosis of the positive slow potential as the result and expression of massive excitation of the neurons of layers IV and III, having followed directly after the arrival of a volley of afferent impulses. It is characteristic that such effects are not observed at deep narcosis and during a poor functional state of the cortex.

(Legend to Fig. 74, textp. 179: Bioelectrical potentials discharged from the surface of the cerebral cortex in the territory of the skin analysor at stimulation of the sciatic nerve. Cat No. 43; surface narcosis. Effects 5 and 6 of recording 58 in enlarged form. In effect, at the first shock of stimulation the positive potential led to a quick fluctuation (as the result of mass excitation of the neurons of layers IV and III), after which a negative potential arose of greater amplitude (expressing regional excitation of the

elements of the surface layers, arising under the influence of

impulses from the neurons of layers IV and III). The second shock

of stimulation caused only a slow positive potential, expressing

regional excitation of the neurons of layers IV and III and arising

under the influence of afferent impulses (Roitbak, 1953b).)

Yet this fact can be interpreted somewhat differently. As

said, excitation of the axons of the motoneurons can proceed even

without the corresponding cells having been stimulated; the axons

can be excited thanks to the stimulating effect of currents governed

by the local cellular potentials (see Chapter I). It is possible

to think that such is the mechanism of excitation of the axons of

the cells of layers III and IV: at the local potential reaching a

certain critical size, the axons of the activated cells begin to

be excited without the local potential having been cut short in

the latter (Fig. 73). From this point of view it is also possible

to explain the fact of the reduction of amplitude and length of the

positive potential in connection with the arising of discharges

of impulses in the axons of the cells of layers III and IV: the

quicker and more vigorously the axons of these cells are excited,

the more quickly and the more intensively will the negative potential

arise; at having arisen, it can completely mask the positive potential.

In Marshall, Talbot, and Ades' paper (1943) the following fact

was reported. Stimulation of the optic nerve of the opposite side
provoked in the area striata a primary response in the form of a
positive potential. If stimulation of the nerve was applied 23.4-58
milliseconds after brief exposure of the ipsilateral eye to light,
that had provoked at the discharge point of the cortex a positive
potential too, then it (the stimulation of the nerve) gave an
entirely altered bioelectrical reaction: instead of a positive
potential, several quick fluctuations of an axonic character arose
and, after them, a strong negative potential. This fact, not subjected
to analysis in their paper, certainly is of the same order as described
above. In the case in question afferent impulses, hitting against
the neurons of layers III and IV in the period of their heightened
excitability and in the period of the beginning in them of local
excitation from the stimulation that preceded, caused the discharge
of these neurons and subsequent spread of impulses of excitation
through the system of ascending axons.

Similar facts have been reported in respect to primary
responses at sound stimulations. In one of Bremer's experiments
sound shocks provoked primary responses limited by the arising of
a positive potential. If 2 shocks were applied with such an interval
that the 2d fell further toward the end of the positive potential
provoked by the 1st, then the effect on the 2d stimulation was

characteristically altered: a negative arose after the positive

potential; thereby the amplitude and length of the initial positive

potential was reduced (Bremer, 1952). In one of Narikashvili's

experiments (1955) sound shocks caused two-phase (+-) primary

responses, which the initial negative fluctuation preceded. At

repeated stimulations the latent period of the arising of the

negative potential was progressively reduced, i.e. the length of the

positive was reduced and, finally, the sound shocks began to cause

purely negative potentials.

It is characteristic that at local strychninization of the

cortex, i.e. at artificial elevation of excitability of the cortical

neurons, the negative potential of the initial responses is intensified

and with this the length and amplitude of the preceding positive

potential is often reduced (Bremer, 1952). It was also observed at

local application of acetylcholine after preliminary atropinization

of the cortex (Chatfield and Purpura, 1954).

Inasmuch as the negative potential arises under the influence

of impulses from the ascending axons and collaterals of the neurons

of layer IV, then on the basis of an analysis of Fig. 74 (effect at

1st shock of stimulation) it is possible to conclude that the

discharge of the neurons of layer IV can proceed upward toward the

surface of the cortex with a very small latent period: in the case

in question the negative potential developed 2-2.5 milliseconds
after the beginning of the positive potential.

Clare and Bishop's data (1954) indicate that association
pyramidal neurons of layer IV are stimulated 2 milliseconds after
the arrival of the afferent impulses. As said, from a small portion
of the cortex within the suprasylvius fissure "secondary" bioelectrical
effects are registered at optical stimulations. In response to
stimulation of the optic nerve, in this region a biopotential arises.
The latent period of its arising is 2 milliseconds greater than the
latent period of the arising of the initial effect in the optic
projection territory of the cortex. A secondary effect in the
suprasylvius furrow arises under the influence of impulses from axons
of the association pyramids of layer IV of the optic projection territory.
The latter are excited under the effect of afferent impulses. Consequently,
the difference in the latent periods of the "primary" and "secondary"
effects (2 milliseconds) is equal, approximately the size of the latent
period of the discharge of the association pyramidal neurons.

It should be noted that at peripheral stimulations responding
cortical potentials arise 2.5 milliseconds after the beginning of the
responding bioelectrical reaction of the cells of the corresponding
thalamic nucleus (Schricker and O'Leary, 1953), i.e. the length of
the "delay" in the neurons of the thalamus is of the order of 2
milliseconds.

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Finally, in the motoneurons of the spinal cord the minimal delay, i.e. the interval of time between the beginning of the local potential and the moment of the discharge of the neurons, equals 2 milliseconds (Eccles, 1946).

Apparently 2 milliseconds is the usual size of the "delay" at spread of excitation from neuron to neuron in the central nervous system of lightly narcotized animal. However, in certain conditions excitation can spread through the central nervous system without marked "delay" (Beritov, 1937a). In case of such quick excitation of the neurons of layer IV (according to type of detonation, as some authors say), from a corresponding point of the surface of the cortex a purely negative potential can be registered in the character of a "primary" bioelectrical reaction of the cortex, since we know that the initial discharge of the cells of layer IV cannot be registered from the surface of the cortex.

As seen in Fig. 74, the 2d shock of stimulation provoked a pure positive slow potential of greater amplitude and length. Bremer and Bonnet (1949) note that when the functional state of the cortex is somewhat impaired (owing to narcosis, asphyxia, drop of blood pressure), then the negative component "of the primary responses" is lacking, but the positive potential can be greatly increased. They give no clarification to this paradoxical phenomenon. These facts

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are easily explained from the point of view of ideas on the mechanism
of the arising of excitation impulses in the neurons of cortical layers
III and IV that are developed in the present chapter. These neurons,
on which the afferent fibers form numerous densely arranged synaptic
endings, are normally extremely easily excited. Under the effect of
a volley of afferent impulses regional excitation arises in them,
which overgrows into a discharge of cells shortly after the beginning
of its development (see Fig. 78, effect at 1st shock of stimulation).
When for one or another reason (deterioration of the functional state,
inhibition) the excitability of the neurons of layers III and IV is
reduced, then under the influence of the same impulses in them a
regional excitation develops in full measure in them and then dies
out (see textp. 78, effect at 2d shock of stimulation).

This explanation agrees with a number of phenomena well known
from the physiology of the peripheral and central nervous system. At
the time of a relative refracting phase of the nerve fiber its electrical
stimulation causes no spreading current of effect, but produces a local
potential of considerably greater amplitude than in ordinary conditions
(Hodgkin, 1938). Braked motoneurons of the spinal cord of cat develop under
the influence of an afferent volley of impulses a considerably greater
local potential than ordinary (Eccles, 1946). Apparently at reduction
of the excitability of the neuron a local potential can reach greater

magnitude without the discharge of the arisen cell having cut short

its course, as would be perceived during the normal aspect of things

(see Bakuradze, Beritov and Roitbak, 1947).

However, during the deepening of narcosis or asphyxia, or in

the course of local poisoning of the cortex, the amplitude of the

positive potentials is progressively reduced in the measure of the

deterioration of the state of the cortex.

Opinions that exist in regard to the origin of the negative potential.

According to one of them, the negative potential, registered from the

surface of the cortex, which a positive potential precedes, expresses

a consequent potential arising after excitation of the neurons of layer

IV (Marshall and coworkers, 1941). Thus, according to Adrian, the

initial positive potential is an expression of the resultant potential

of afferent fibers. According to Marshall, the negative potential is

the resultant potential of neurons of layer IV. From this point of

view it is extremely difficult to clarify a whole series of facts.

Why, for instance, is the negative phase of the "primary responses" as

a result of surface thermocoagulation of the cortex selectively depressed?

Or why is the territory from which the negative potentials are discharged

larger than the territory from which the initial positive potentials are

discharged?

The most widespread point of view, according to which the negative

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potential arises as the result of excitation spread from the cellular

bodies of the pyramidal neurons of layer IV upward through their

dendrites into layer I (Chang and Kaada, 1950; Chang, 1953; Bishop

and Clare, 1953), is the following: excitation arising under the

influence of the afferent impulses in the body of the pyramidal

neuron at the level of layer IV is registered from the cortical

surface as a positive potential; when, spreading then through a

dendrite, it approaches the discharge electrode, a negative potential

is registered. According to Bishop, in order for an ascending

dendrite of a pyramidal neuron to be excited an exciting influence

is necessary on the body of the cell and on the basal dendrites

by a great number of synaptic endings; otherwise, the excitation

does not spread upward, and from the surface a one-phase (positive)

potential is registered.

It has already been said that, according to Chang, the top

dendrites of the pyramidal neurons conduct excitation like the nerve

fiber in both directions, i.e. both from the body of the pyramidal

neuron upward to the ramifications of the top dendrite, and also

from the branchings of the dendrites in layer I downward to the body

of the pyramidal neuron. Bishop came to the conclusion that the

top dendrites of the pyramidal neurons are devoid of the capacity

to conduct excitation downward to the body of the cell (see Bishop
and Clare's 1955 article), but they have the capacity to conduct it
in the opposite direction, i.e. from the body of a pyramidal neuron
through a top dendrite upward to layer I. In other words, when the
top dendrites are activated through the synaptic endings located on
them, they are not obliged to conduct excitation; when the body of
the neuron is excited, then these dendrites conduct excitation in
the antidromic direction.

According to Artem'ev's point of view (1951), the positive
potential and negative potential are connected by the activity of
one and the same nerve elements located in the surface portions of
the cortex.

The main facts on which the conclusion is based were obtained
in experiments with polarization of the auditory territory of the
cortex. With a current intensity of 0.1-0.3 milliampere and when
the anode is found in the auditory projection territory, the positive
phase of the primary response is reduced or absent and negative
phase sets in. This, according to Artem'ev, occurs "as the result
of blocking, by a polarizing current, of the nerve processes
responsible for the development of the positive phase". The same
processes which produce the second, the negative phase, are not
provoked. At cathodization of the auditory territory of the cortex

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an opposite phenomenon was observed, the presence of a positive phase
and reduction, sometimes even the absence, of the negative phase.
The same results were obtained by Goldring and O'Leary (1951) in
experiments on area striata.

These facts can receive an entirely different explanation from
that which Artem'ev gave them. They do not at all conflict with the
ideas developed in this chapter, according to which the positive and
the negative potential express regional excitation of neuronic elements
of different layers of the cortex, namely IV and I. A negative
potential arises under the effect of impulses from the excited cells
of layer IV, etc. At analysis of the recordings which Artem'ev
and which Goldring and O'Leary cite, it is possible to note a series
of characteristic factors. 1) At anodization of the cortex, usually
no complete disappearance of the positive potential occurred: either
it was greatly attenuated or it expressed one or two quick fluctuations
(Artem'ev, 1951, Fig. 2, D and E). 2) The duration of the positive
potential was reduced, and the negative potential set in now too
without pause after the positive, i.e. the latent period of the arising
of the negative potential was reduced and, when the positive was
represented by a quick fluctuation, the impression was created that
the negative potential set in directly in response to stimulation, i.e.
that the positive potential inverted its sign. 3) The amplitude of

the negative potential at the time of anodization of the cortex was

increased (Goldring and O'Leary, 1951).

Thus, under the influence of polarization of the cortex a picture

arises that is already familiar to us, presented in Fig. 74, in which

the second shock of stimulation of the sciatic nerve provoked in the

cortex a positive potential of greater amplitude and duration, and

the first was an initial, quick, positive fluctuation (which at a slow

rate of exposure can generally disappear), after which a powerful

negative potential follows. The change of cortical potentials described

is evidently explained in the following way. As Goldring and O'Leary

indicate, at anodization of the surface of the cortex the top dendrites

of the pyramids are anodically polarized, but the bodies and axons of

the corresponding neurons undergo cathodic depolarization, i.e. the

same electrical field arises as at local excitation of the neuronic

elements in the deep layers. Thus, as a result of anodization of the

cortical surface a cathodic elevation of excitability of the neurons

of layer IV and facilitation of their excitation under the effect of

afferent impulses should occur. This is expressed oscillographically

in a reduction of the length and amplitude of the initial positive

potential and in the intensification and acceleration of the moment

of the arising of the negative potential. At cathodization of the

surface of the cortex the negative potential ceases to be provoked,

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but the positive is intensified and its length is increased:

cathodization of the surface of the cortex leads to anodic polarization

of the elements of layer IV, to reduction of their excitability. These

elements react on the afferent impulses with regional, nonspreading

excitation (see textpages 56, 57, and 131).

Thus, when from the surface of the cortex potentials of different

signs are discharged, this indicates that in these cases excitation

occurs of the different neuronic elements located in the different layers

of the cortex.

Contribution to the question of the spread of bioelectrical

potentials through the cortex at peripheral stimulations. We have

seen that primary responses are registered from strictly limited

parts of the cortex; for example, in response to stimulation of the

sciatic nerve (Fig. 62) or in response to the effect of sound shocks

(Fig. 70) they arise in the skin and auditory projection territories,

respectively, but are not observed in other parts of the cortex.

Bremer (1943), Boyarsky and Peacock (1952), and many other authors

also observed this. We have seen, moreover, that even within the

limits of any projection territory whatsoever of the cortex in

response to various stimulations of a given character mainly one or

another part of this territory is activated. For instance, different

points of the gyrus ectosylvius med. of dog are excited mainly at the

effect of tones of a certain height (Tunturi). It was mentioned

higher up that in cat there is no such kind of strict localization

of sounds in the auditory territory of the cortex, but that this

is not stipulated by irradiation of excitation from the irritated

point to the whole of the auditory territory. However, Mickle and

Ades (1953) came to the conclusion that the "primary response" of

the auditory territory of the cortex in cat, arising first at a

certain point of it, spreads then radially at the rate of 1.5 meters

per second to the whole auditory territory. This conclusion was

made on this basis, that the latent period of the primary response

is increased in the measure of removal from the part of greatest

activity (the part in which, at response to sound shock, the

answering biopotentials of greatest amplitude are registered).

  Adey, Carter, and Porter (1954), who studied primary responses

arising in the skin-muscle projection territory at electrical

stimulation of subcutaneous tissues (en masse) of an extremity,

obtained similar facts. It was demonstrated that the latent period

of the primary responses is different at different points of the

corresponding portion of the cortex and that there is a definite

relationship between the size of the latent period and the amplitude

of the biopotentials: effects of greater amplitude had a lesser

latent period. For instance, at stimulation of the opposite hind

paw a portion of the cortex 7 x 2 mm. in size was activated; at its

rear boundary the latent period of effects equaled 20 milliseconds;

at a point 2 mm. toward the front the latent period equaled 14

milliseconds; at the front boundary it was again increased to 20

milliseconds. At stimulation of a homolateral extremity the latent

period of effects at different points of this portion of the cortex

was identical.

Marshall, Woolsey, and Bard (1941) found that the latent

period of primary responses at different points of the skin projection

territory can be diverse (fluctuations of from 13.5 to 16 milliseconds).

With the least latent period of 13.5 milliseconds, effects arose at

that point where they had a maximal amplitude. From their recordings

it is possible to conclude that, according to the measure of removal

from this point, the latent period of the primary responses was

successively increased (13.5 - 14 - 14.5 - 16 milliseconds).

Is it correct, however, on the basis of these facts to conclude

on the irradiation of primary responses through the cortex, i.e. of

the complex known to us, which consists of the initial positive slow

potential, after which a negative slow potential may follow? Is it

possible, on the basis of the different latent periods of their arising

at adjacent points of the cortex, to conclude that the process arising

locally under the effect of the afferent impulses then spreads from

neuron to neuron at a certain rate? The phenomenon described, i.e.

the arising of primary responses with diverse latent period at points

of the cortex lying in a line, is also observed when at deep narcosis

the reaction is limited by the arising of purely positive potentials.

To recognize the irradiation of these potentials through the cortex in

these cases means to recognize an inaccuracy of the theoretical

conclusions made above in regard to the origin and significance of

the positive potentials of the primary responses. Actually then

regional nonspreading excitation that does not provoke discharges of

impulses to the axons cannot stipulate a subsequent involvement of

surrounding neuronic elements in this same process. This phenomenon

is certainly specified by other circumstances. Already in each sensory

thalamic nucleus where afferent impulses come at corresponding peripheral

stimulations there is a definite differentiation. In the first place,

the system of thalamocortical projection fibers is organized so that

the different thalamic cells of a given nucleus are connected mainly

with the different points of the corresponding projection territory

of the cortex. In the second place, a given group of afferent fibers

is connected more closely with a certain group of thalamic cells,

but those fibers with thalamic cells lying in a line are connected

less closely, i.e. they form a smaller number of endings on them (see

Galambos, 1954). When excitation comes to the thalamus along the

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fibers in question, then primarily the thalamic neurons with which
these fibers are mainly connected are stimulated by impulses and the
neurons lying in line begin to be stimulated later, since regional
excitation in them later reaches the critical magnitude at which
they begin to be stimulated. Hence, afferent volleys of impulses
from the thalamus come to different points of the cortex at a different
time. As a result, at peripheral stimulation a focus of greatest
activity arises in the cortex, surrounded by a territory where the
neurons are activated not only later but also more slightly. For
instance, at stimulation of a muscle nerve on the paw, in a certain
part of the skin-muscle projection territory of the cortex there is
a "focus" from which answering biopotentials of greatest amplitude
are discharged. If the electrode is shifted 0.5-1 mm., the amplitude
of the potentials discharged from a point adjacent to this proves
highly attenuated (Gardner and Haddad, 1953).

From this point of view the fact is readily explained that at
worsening of the functional state of a preparation (for instance, at
a drop of body temperature) narrowing occurs of the territory of
primary responses to sound shocks (Pribram and coworkers, 1954):
first and foremost those thalamic neurons which are more weakly
stimulated at a given peripheral stimulation cease to be stimulated
by impulses to the cortex.

At peripheral stimulation some foci of maximal bioelectrical activity arise in the cortex; for instance, at stimulation of part of the skin of cat a focus arises in the gyrus sigmoideus post, and a focus arises in the territory of the gyrus ectosylvius ant. Each focus occupies an area less than 1 mm^2 and is surrounded by a territory from which potentials of lesser amplitude are discharged. The breadth of this territory is 1-2 mm. (in monkeys it is up to 4 mm.). The arising of several foci of maximal bioelectrical activity in response to peripheral stimulation is not stipulated by spread of excitation from one part of the cortex to another. Each focus is an independent projection from the thalamus (Marshall and coworkers, 1941).

Thus, in response to a given peripheral stimulation in the cortex there arises, in a corresponding projection territory, a focus of maximal excitation. It can be ascertained in narcotized animals on this basis, that primary responses (positive slow potentials) arise in it with a minimal latent period and have greatest amplitude.

The physiological importance of a negative potential of primary response. We have explained that a positive potential of primary response reflects regional excitation of the neurons of layers IV and III. When regional excitation reaches a certain critical magnitude, a discharge of impulses of excitation arises from these neurons.

Excitation of the neurons with ascending axons spreads upward to the
surface of the cortex and stipulates the arising of a negative potential,
reflecting regional excitation of the top dendrites. What is the
physiological importance of this bioelectrical reaction?

At excitation of the neurons of layer IV impulses proceed not
only upward to the surface of the cortex, but downward into the system
of efferent fibers, as well as horizontally through association fibers
to certain other parts of the cortex. The more powerful the discharge
into the association and efferent fibers, the more powerful the
discharge to the surface of the cortex.

These conclusions issue from the following series of facts.

1. In response to sound stimulations primary responses arise
in the auditory projection territory; but certain fluctuations of
potential arise too in certain other territories of the cortex, for
instance in the optic (in the gyr. splenialis). These fluctuations
at the time of the beginning of their development agree with the
appearance in the auditory projection territory of a negative phase
of the primary response (Artem'ev, 1951).

2. As said, in response to sound stimulations a bioelectrical
reaction arises in the lower part of the gyrus ectosylvius post.
because of the transmission from here of impulses from the primarily
excited auditory projection territory. After local strychnine

poisoning of the gyrus ectosylvius med., side by side with extraordinary

intensification of the negative phase of the first response, the

biopotential in the gyrus ectosylvius post. is also intensified; at

times it arises in conformity with the negative phase of the primary

response (Bremer, 1952; Bremer and coworkers, 1954).

     3. Stimulation of the n. medianus provokes primary responses

in the skin projection territory of the cortex. When a negative

potential of the primary response is weakly expressed, then in the

motor territory of the cortex no bioelectrical reaction arises at

all. When, owing to the artificial elevation of excitability in a

corresponding part of the skin projection territory, stimulation of

the n. medianus begins to provoke primary responses, in which the

negative phase is considerably intensified, then this is associated

with the arising of a bioelectrical reaction in the motor area of

the fore paw: fluctuation of potential in the motor area sometimes

coincides with the negative phase of the primary response in the

skin territory (Chatfield and Purpura, 1954).

     4. When in response to peripheral stimulations from the cortex

only positive potentials are discharged, then no changes occur of the

"spontaneous" electrical activity either in that territory where these

potentials are registered or in other territories of the cortex. When

peripheral stimulations provoke in the cortex complex bioelectrical

reactions, then they thereby influence too the "spontaneous" electrical

activity, the changes of which can be of a diverse character depending

on the kind of narcosis and the degree of its depth (Adrian, 1941).

Apparently this reaction too is the result of the spread of excitation

along the cortex from a point of the cortex excited by afferent

impulses (see below).

     5. It is known that at arrival of afferent impulses in the

sensory-motor territory of the cortex excitation is transmitted by

means of the neurons of layer IV to efferent pyramidal neurons of layer

V. This reflex activity is also preserved during narcosis. At

discharge of potentials from the cortical surface in the territory of

the skin analysor and simultaneously from the fibers of the pyramidal

pathway, in response to adequate stimulation of the corresponding part

of the cortex, the following bioelectrical reaction is registered (see

Fig. 75): after the usual latent period an initial positive potential

arises, after it a negative potential arises and simultaneously a

discharge of impulses forms in the fibers of the pyramidal pathway.

When stimulation provokes a reaction limiting the arising of only

the initial positive potential, then a discharge of impulses does not

arise in the fibers of the pyramidal pathway. When there is an absence

of discharge, the positive potential has greater amplitude and length.

In Fig. 75 it is seen that the more powerful the discharge into the

pyramidal pathways, the more powerful the negative potential discharged
from the surface of the cortex; hence, it is possible to conclude that
the more impulses proceed into the fibers of the pyramidal pathway
the more of them proceed along the ascending axons and collaterals into
layer I* and the more intensive the activation of the top dendrites of
layer I in the portion first activated by afferent impulses. (*In the
case in question apparently not only from the recurrent collaterals
of the pyramidal neurons of layer IV but also from the recurrent
collaterals of the pyramidal neurons of layer V.)

     (Legend to Fig. 75, textp. 188: Discharge of impulses into
pyramidal pathways, associated with the negative potential of the
primary response. Cat under chloralose narcosis. Upper curve - from
surface of sensory field for a front paw, lower curve from a needle
electrode in the territory of the crossing of pyramidal pathways.
Effect of four touches on the paw. At the first two stimulations
only positive potentials arose, at the third and fourth a negative
potential followed the positive, and these effects were associated
with discharges of impulses in the pyramid pathways. (Adrian, 1941).)

     Beritov, on the basis of analysis of histological and
physiological data, concluded that association neurons, transmitting
excitation to more or less remote points of the cortex, simultaneously
by means of their collaterals should also affect neurons closely adjacent

and that they probably depress these cells by activation of their
dendrites, by which localization of excitation is guaranteed in its
initial part (Beritashvili, 1953).

From Chang's data it is possible to conclude that under the
influence of a negative potential, provoked by stimulation of the
surface of the cortex, inhibition occurs of the neurons of layer III:
the negative potential of the callosal effect, i.e. of the effect
provoked at this point by stimulation of a symmetrical point of the
other hemisphere, falls out. At the present time there are facts
indicating that under the influence of a slow negative potential,
provoked by tetanizing stimulation of the cortical surface, inhibition
occurs of the neurons of layer IV (Beritov and Roitbak, 1955).

The territory of the arising and registration of the negative
potentials being considered is limited by the territory of the surface
layers of the cortex on which the synaptic endings of the ascending
axons and of the collaterals of the neurons of layer IV are spread;
the ramifications of these axons and collaterals in layer I, comprising
the main component of the system of fibers of layer I (see Chapter II),
evidently do not proceed for considerable distances. However, it is
characteristic that the negative potential is registered over a larger
territory than the initial positive one (Morison and Dempsey, 1942).
This is entirely conceivable, proceeding from the fact that the

ascending axons and collaterals run a certain distance in layer I
and activate the top dendrites over a somewhat larger territory than
that where activation of the primary focus in layer IV occurs under
the influence of the afferent impulses.

Proceeding from all that has been said above, it is possible
to conclude that at arrival in the cortex of a volley of afferent
impulses a focus of excitation arises surrounded by an aureole of
inhibition.

Contribution to the question of sensory cells scattered through
the cortex. According to Pavlov, each receptor apparatus has in the
cortex a special individual central territory, which represents its
distinct projection. Here, because of the peculiarities of construction
(dense distribution of cells, their most numerous connections, etc.),
highest analysis and synthesis are accomplished. However, the cortical
sensory cells in question, spread too beyond the limits of this territory,
can be over the whole cortex, now being arranged ever less densely
according to the measure of removal from the nucleus of the analysor.
A more elementary and rough analysis of the peripheral stimulations is
connected with these scattered elements (Pavlov, 1922; see also
Pavlovskie sredy, I: 137, 214, 222).

As seen from factually presented material and from a survey of
existing data, primary responses arise in limited areas of the cortex.

Finally, on the basis of data obtained in the pointed conditions
of the experiments, it would be impossible to make definite conclusions.
Nevertheless, recently it has been clarified that in non-narcotized
cats sound stimulations provoke characteristic direct bioelectrical
effects (primary responses), which set in with a latent period of about
10 milliseconds and have an initial positive slow component. They
arise also at time of stimulated state and in the state of natural
sleep, and, what should properly interest us at once, these primary
responses arise in normal animals in a limited portion of the cortex,
in the very part in which they are registered in pointed experiments
(Roitbak, 1954b). The same was ascertained in regard to primary
responses at cutaneous stimulation (Roitbak, 1954c and 1955b). In
people from the exposed cortex (at time of operation under local
anesthesia), in response to stimulation of the skin, primary responses
were registered in the region of the gyrus centralis post. like those
which arise in animals (Marshall and Walker, 1949). With the use of
a definite technique of taking down the EEG in man through intact
skull primary responses were registered to optical stimulations
(Monnier, 1952) and to electrical stimulation of cutaneous nerves
(Dawson, 1947, 1954a; Larson, 1953). In response to exposure of the
eye to light, response biopotentials arose in the occipital region,
and in response to stimulation of the cutaneous nerves biopotentials arose
in the region of the gyrus centralis post.

Thus, in normal animals and in man a given peripheral stimulation provokes direct bioelectrical effects (primary responses) in a limited area of the cortex, in the corresponding projection territory, and does not provoke them in other parts of the cortex.

In pointed experiments one other very conclusive fact was ascertained: primary responses to a given peripheral stimulation arise in the corresponding projection territory, and they do not arise in other territories even at strychnine poisoning of the latter when the cortical neurons, as we shall see later, are excited under the influence of a minimal "impulsation" that does not appear in ordinary conditions on the electrocorticogram. Furthermore, the stimulation in question causes primary responses only in a certain part of the corresponding projection territory: as said, in dog the gyrus ectosylvius med. is connected with different parts of the membr. basilaris; i.e., at the effect of a definite tone, excitation occurs of neuronic elements of a certain part of the convolution; at the effect of a remote tone, this part is not excited even during its local strychnine poisoning (Tunturi, 1950).

Oscillographic data confirm, as we have already said, the position that the cells of perception of the cortex are arranged most densely in the central part of the analysor and, according to the measure of removal toward the periphery, the density of distribution

of the cells is reduced. However, oscillographic data give no

indications that the cells of perception[*] of each analysor are scattered

throughout the whole cortex and that, thus, in the territory of each

analysor to one or another degree all other analysors are represented.

([*]Those cells are intended on which afferent fibers from the differenti-

ated sensory nuclei of the thalamus terminate directly: from the

corpus geniculatum laterale, the corpus geniculatum mediale, etc.)

Popov too came to a like conclusion on the basis of experiments with

extirpations in some cases of the whole cortex and in others of the

cortical ends of the separate analysors. After removal of the cortical

end, for example, of the sound analysor, the conditioned reflexes to

sound stimulations that were present disappeared. All positive and

negative conditioned reflexes have been fully preserved with intact

analysors. After several hundred combinations of sound stimulation

with electrical stimulation of the skin, primitive motor reaction

succeeded in being obtained, but this reflex was not differentiated

and did not extinguish. The fact of the elaboration of this reaction

cannot serve as demonstration of the presence of cells of perception

strewn through the cortex: in dog after removal of the entire cortex

precisely the same primitive reaction, of clearly subcortical origin,

can be produced (Popov, 1953).

The concept of the scattered elements has no morphological bases,

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i.e. there are no data on the fact that from a given sensory thalamic
nucleus (for example, the corpus geniculatum mediale) projection
fibers proceed into all areas of the cortex.

Oscillographic data confirm the opinion that not all the
cerebral cortex is occupied by special projection territories and
that free territory is left. In rabbit it is very small, i.e. almost
the whole cortex is occupied by fields in which primary responses are
registered at corresponding peripheral stimulations. In cats this
territory is quite vast and includes, besides the other territories
of the cortex, almost the whole gyrus suprasylvius med. and part of
the gyr. ectosylvius post. (Fig. 55). In monkeys it is considerably
larger than in cats (Rose and Woolsey, 1949).

The results of oscillographic investigations permit concluding
that these territories of the cortex are not activated directly at
stimulations of any receptors whatsoever. As said, in certain of
these territories secondary bioelectrical reactions arise from the
projection territories of the cortex first excited. However, existing
electrographical data give no opportunity to judge on the functions
of these "free" parts of the cortex.

All the same, the question of the sensory cells scattered
throughout the cortex cannot possibly be considered conclusively
resolved. Perhaps the primary responses are not registered within

the limits of the corresponding projection territory because there

is very little density of their arrangement and biopotentials

generated by these individual cells are not intercepted. Perhaps

sensory cells in other territories of the cortex are represented

only by neurons with short axon, having no dendrites that ascend to

the surface of the cortex and, because of this circumstance, their

electrical activity cannot be detected at discharge from the surface

of the cortex. Apparently special investigations are needed to

solve this question.

2. Contribution to the Question of Generalized Reactions of the Cerebral Cortex

Concerning the nature of strychnine convulsive potentials.

As well known, at local strychnine poisoning of the cortex a certain

time after application of the strychnine so-called strychnine bio-

electrical potentials arise of characteristic configuration: after

a relatively small positive fluctuation a negative fluctuation

develops of greater amplitude, after which a long positive oscillation

follows. According to the measure of development of the poisoning,

convulsive potentials set in ever more frequently, and sometimes their

correct rhythm is ascertained. If the poison continues to lie (there),

then convulsive activity can be observed for a period of an hour or

more. After removal of the poison, the bioelectrical activity of

the poisoned part gradually (after 30 minutes and more) returns to

the initial state. Convulsive potentials arising at local-strychnine
poisoning of the cortex reach an amplitude of up to 5 milliv. In
spite of this, they are not registered from adjacent unpoisoned
territories of the cortex and also from points of the cortex found
2-3 mm. distant from the part poisoned (Bartley, O'Leary, and Bishop,
1937; Beritov and Gedevanishvili, 1945). This is one more good proof
that in the cortex the bioelectrical potentials are not spread in a
purely physical way to any considerable degree.

The local character of the convulsive potentials, i.e. the
fact that the territory of their arising is limited by the territory
of the poisoned part of the cortex, depends on two circumstances.
First, it depends on the fact that the poison at local poisoning
of the cortex does not spread to any considerable degree to adjacent
cortical territories. Lying at the base of this is the peculiar
distribution of the blood vessels in the cortex. At each given
point of the cortex, from the artery of the pia mater an intracerebral
artery goes out, which proceeds in a vertical direction deep into
the cortex, breaks down into capillaries that pass over into capillaries
proceeding near a vein which emerges at the surface of the brain in
the venous network of the pia mater (Klosovskii, 1952). At application
to the surface of the cortex of a sheet of filter paper or a ball of
cotton saturated with strychnine, quick poisoning occurs of the whole

thickness of the cortex at the point of poisoning. Thereby the

elements of all layers are poisoned approximately simultaneously

although it has been observed that the surface layers are poisoned

earlier (Bartley and coworkers, 1937; Beritov and Gedevanishvili,

1945). Since circulation of the blood is accomplished by blood

vessels proceeding vertically through the cortex, then the

strychnine is quickly absorbed by the part of the cortex which

is supplied with blood from those vessels and is secured simply

by the cortical cells; the excess of poison is so diluted in the

veins that it shows no marked effect on the remaining substance

of the cortex (Bartley and coworkers, 1937). Secondly, the local

character of the convulsive potentials is stipulated by the fact

that intense nerve activity of convulsive character in one part

of the cortex does not involve in this activity the adjacent parts

of the cortex, i.e. no spread of excitation occurs from the poisoned

neurons to adjacent nonpoisoned neurons. In Beritov's opinion,

the very intense nervous activity in the poisoned part of the

cortex itself creates a condition that impedes its spread through

the cortex. This condition is the inhibition of the adjacent complexes of

neurons (Beritov and Gedevanishvili, 1945; Beritov, 1948). A like

explanation was given for the fact that at tetanic stimulation of the

cortex a limited focus is created of most intensive bioelectrical

activity, i.e. no spread of it occurs to the remaining cortex (see
Chapter III). Lack of spread of the convulsive activity from the
poisoned part of the cortex is not connected with narcosis, i.e.
with reduced excitability of the cortex. This is demonstrated by
the following fact: in non-narcotized dog at local strychnine
poisoning (1% solution) of part of the motor territory of the cortex
energetic shudders of the corresponding muscles set in, but they
do not spread to other muscles whose centers lie in direct proximity
with the poisoned part (Amantea, 1912).

The strychnine potentials are frequently designated "spontaneous"
convulsive discharges, since they begin to arise as though without
connection with any appreciable outer or inner stimulation; however,
at the present time the very fact of the presence of "spontaneous"
electrical activity in general is under question. Burns (1949, 1951)
ascertained that neuronic elements of isolated strip of cortex with
the circulation preserved do not generate, at lack of narcosis,
"spontaneous" bioelectrical potentials[*]; at the same time one shock
of electrical stimulation applied to the surface of this strip of
cortex provokes a powerful, complex, and prolonged (up to 4 seconds)
electrical reaction (Fig. 29). ([*]However, according to the data
of Kristiansen and Courtois (1949), several hours after operation
the isolated portion of cortex begins to produce a series of "spontaneous"

biopotentials. According to Burns, if spontaneous activity is

observed in the isolated portion of cortex, then this is always

connected with injury of the cortex and is stipulated by irradiation

of excitation from the injured point; after thermocoagulation of

this point the electrical activity disappears (Burns, 1951).)

Isolated strip of cortex after narcotization does not disclose,

as would be expected, "spontaneous" electrical activity. In

response to a single electrical stimulation, it produces a simple

brief (0.1 second) electrical reaction. It is known, furthermore,

that in ordinary experiments on narcotized animals a single

stimulation of the cortex produces the same simple bioelectrical

reaction; but uninterrupted "spontaneous" electrical activity,

particularly alpha-rhythm fluctuations, discharge from the cortex.

On the basis of the above-mentioned facts, it is possible

to conclude that in Burns' experiments the absence of "spontaneous"

electrical activity in isolated strip of cortex was not connected

with poor functional condition of this portion of the cortex, since

tests with direct electrical stimulation showed high excitability

and the great functional possibilities of isolated non-narcotized

strip of cortex as compared with uninjured cortex of narcotized

animal. At the same time the latter produces spontaneous fluctuations

of potential. It is characteristic that if between isolated strip of

cortex and the remaining cortex a narrow bridge of cortex was
preserved, then the strip of cortex generated the usual "spontaneous"
fluctuations of biopotential (Burns, 1949).

Evidently "spontaneous" electrical activity of the cortex is
stipulated by afferent impulses (afferent in the sense of their
coming to the cortical neurons in question) and is not an expression
of the fundamental property of the cortical neurons to be excited
automatically, "to be self-excited" at a certain rhythm, as Bremer
(1949) thinks, and is not stipulated by the stimulating action on
them of different chemical substances (hormones, metabolites,
electrolites) that arise within the organism, as Beritov assumed
(1947).

Below are presented facts obtained in the course of an analysis
of convulsive strychnine potentials, which can serve as argument in
favor of the so-called reflex origin of "spontaneous" bioelectrical
potentials in the cerebral cortex.

The strychnine potential in the cortex is associated with
efferent discharge of impulses of excitation into the white matter
(Adrian, 1941). First, from the poisoned part impulses proceed into
the callosal fibers, as a result of which a certain bioelectrical
reaction arises at a symmetrical point of the opposite hemisphere.
Secondly, impulses spread from the poisoned part through association

fibers, stipulating secondary activation of certain, often remote,

parts of the cortex. (On this is based the so-called strychnine

neuronographia, i.e. one of the methods of determining the anatomical

connections within the cortex.) Finally, through projection fibers

impulses from the poisoned part spread downward into the subcortical

formations through the pyramids (Adrian and Moruzzi, 1939; Arduini

and Whitlock, 1953) and through the extrapyramidal courses. For

instance, at strychnine poisoning of the 4s field of the cortex in

the reticular formation of the medulla oblongata, synchronously with

the strychnine potentials in the cortex discharges of impulses are

registered which spread, as has been demonstrated, through the

extrapyramidal fibers (McCulloch, Graf and Magoun, 1946). The same

was observed at local strychnine poisoning of the motor territory

of the cortex, and it has been shown that impulses from the strychninized

part spread into the reticular formation of the medulla oblongata

through the pyramidal courses (Baumgarten, Mollica, and Moruzzi, 1954).

Thus, at time of convulsive discharge all types of pyramidal

neurons of the poisoned part are excited.

At general strychnine poisoning of almost all efferent nerves,

except the vagus, discharges of impulses are registered that coincide

with the convulsive potentials in the central nervous system (Frankenhaeuser,

1951). At local strychnine poisoning of a certain part of the motor

territory of the cortex, as already said, discharges of impulses
should arise only in those efferent nerves which innervate the
group of muscles the center of which was poisoned. The strychnine
potential in the cortex (Bremer, 1936) corresponds to each muscular
twitch, but the convulsive potential in the motor region of the
cortex is not always attended by contraction of the corresponding
muscles: at relatively light poisoning convulsive potentials can
arise for some time without motor phenomenon (Beritov and
Gedevanishvili, 1945).

At local strychnine poisoning of part of the motor territory
of the cortex, for instance of an anterior extremity, after a certain
time rhythmical shudders of the animal's contralateral extremity
begin. This phenomenon has been described by Amantea (1912), and
Beritov, subjecting it to myographic analysis, also clarified that
the movement of a paw (or leg) bears a coordinated character, i.e.
the flexor muscle contracts and the extensor is inhibited at this
time. He considered these twitches the result of internal or external
stimulations provoking an effect at a focus of excitability heightened
under the influence of strychnine (Beritov, 1917). In experiments on
cats under nembutal narcosis we succeeded in establishing that in
certain conditions twitches of an extremity after poisoning of a
corresponding part of the cortex set in precisely at the rhythm of

respiration. This fact shows that impulses arising during the
respiratory act are capable of stipulating excitation of the
pyramidal cells of the cortex, giving rise to pyramidal routes.
Furthermore, it was successfully established that in certain
conditions the convulsive potentials in the poisoned part of the
cortex set in precisely at the rhythm of the respiration (Fig. 76,
textp. 196: Convulsive bioelectrical potentials in the cortex
at the rhythm of respiration. A - cat under deep nembutal narcosis;
potentials are discharged from a point of the gyrus suprasylvius
poisoned with 1% strychnine; respiratory movements of the chest
are registered, and rise of the curve indicates inspiration. B
and C - cat under light nembutal narcosis; the potentials are
discharged from a point of the gyrus suprasylvius poisoned with
1% strychnine, 1 min. after application of the poison to the cortex;
fluctuations are registered of air pressure in the tracheal tube,
rise of curve (indicating) inspiration. D - cat under deep nembutal
narcosis; the potentials are discharged from a point of the gyrus
ectosylvius poisoned with 1% strychnine; respiratory movements of
the chest are registered. E - non-narcotized rabbit with the cerebral
hemispheres revealed; potentials are discharged from a poisoned (1%
strychnine) part of the pariental territory (Roitbak, 1953c).) (It
might be assumed that the phenomenon observed is the result of the

following circumstance. The brain pulses with the respiration;
the discharge electrode found on the surface of the poisoned part
mechanically stimulates the cortical elements, and a bioelectrical
discharge arises corresponding to each respiration. The results of
control experiments have permitted rejecting this hypothesis.

o   Experiments were made with very fine electrodes, which could not
stimulate the brain, and with microelectrodes inserted into the
cortex, which moved together with the brain at its visible and
invisible pulsations. Experiments were also made in which the
respiratory pulsation of the brain, etc., was deliberately intensified.)
This phenomenon can be observed at local strychnine poisoning of
any part of the cortex. Convulsive bioelectrical potentials arose only
in the area of the poisoning: They were not discharged from the
adjacent parts (Fig. 78, textp. 198: Bioelectrical potentials arising
in connection with respiration and in connection with stimulation of
the sciatic nerve in a portion of the skin analysor that had been
poisoned with strychnine. In recordings A-C the potentials are discharged
from a portion of the skin analysor poisoned with 1% strychnine (upper
curves) and from the pariental area. A - convulsive discharges arising
in connection with each respiratory movement. B - beginning of brief
stimulation of the sciatic nerve of the opposite side; frequency of
stimulation 3 per second; intensity 1.5 v.; vertical lines before the

bioelectrical effects denote the moment of stimulation. C - end of

stimulation of the sciatic nerve; convulsive discharges arose in

connection with alternate respiratory movement. In recordings D

and E potentials are discharged from the poisoned part. D - end

of prolonged stimulation of the sciatic nerve. E - 0.5 min. after

D; resumption of convulsive discharges. All the experiments were

produced on one preparation (cat No. 43). A recording of the

respiration underneath on all the oscillograms; rise of curve

indicates expiration. (Roitbak, 1953c).)

Convulsive discharges appear 0.5-3 minutes after the moment of

poisoning (1% solution of strychnine). At first they are irregular

and infrequent, then they increase in frequency and a period sets in

when they follow the rhythm of the respiratory movements of the chest,

arising either in the phase of inspiration or in the phase of

expiration. A single convulsive potential in the cortex, or a group

of 2-3 or more potentials, can correspond to each respiratory movement.

Convulsive discharges arising in the phase of inspiration can, after

a certain time, begin to arise in conformity with each expiration (Fig.

76, B and C), and contrariwise. With different conditions, with a

different concentration of strychnine, a different depth of narcosis,

or in another stage of poisoning, it is possible to observe the

arising of convulsive potentials both in the phase of inspiration and

in the phase of expiration, i.e. their frequency doubles. If thereby

convulsive potentials arise by groups, then at times their almost

uninterrupted rhythm is ascertained and, in such cases, it is difficult

to determine the connection of their arising with the respiration.

Finally, the rhythm of the convulsive discharges can be more infrequent

than the rhythm of the respiration, for example convulsive discharges

can arise at each second respiratory movement of the chest.

The phenomenon considered, the arising of convulsive potentials

at the rhythm of the respiration, can be observed too at local

strychninization of the cortex of non-narcotized animal (Fig. 76, E).

It is known that if a discharge electrode is established on the

surface of the cortex in the territory of the nucleus of one or

another analysor, then at stimulation of the corresponding sensory

nerve or of the corresponding receptors characteristic bioelectrical

effects are registered. In answer to each single stimulation, with

a latent period of 8-15 milliseconds, a positive fluctuation of

potential arises; a negative fluctuation can follow after it. After

local strychnine poisoning of the cortex the negative fluctuation

intensifies, after which a considerable positive fluctuation now

follows. Thus, under the influence of strychnine the general character

of the primary effect arising in response to the corresponding peripheral

stimulation is not altered.

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In Figs. 77 and 78 are presented effects arising, at stimulation
of the sciatic nerve, in a poisoned part of the cortex behind the
cruciate sulcus (portion of the nucleus of the skin analysor correspond-
ing to the projection for the skin of a rear extremity). During
stimulation at a rhythm of 2 or 3 per second, in response to each
shock of stimulation the effect described above arises: an initial
positive fluctuation, after which a negative fluctuation of greater
amplitude follows. The so-called "spontaneous" convulsive potentials
registered from the part of the cortex poisoned with strychnine are
entirely similar in character to the potentials just described, which
arise in response to peripheral stimulations. This circumstance
has been noted by a series of authors (Bartley, O'Leary, and Bishop,
1937; Chang and Kaada, 1950). However, as far as known to me, the
following guiding conclusion has not been made by anyone: the
convulsive strychnine potentials are stipulated by some afferent
impulses proceeding in the cortex. (Legend to Fig. 77, textp. 197:
Bioelectrical potentials arising "spontaneously" and in connection
with stimulation of the sciatic nerve in a part of the skin analysor
poisoned by strychnine. A - cat No. 13; the potentials are discharged
from a portion of the gyrus sigmoideus post. poisoned by strychnine
(0.1% solution). The sciatic nerve of the opposite side is stimulated.
Frequency of stimulation 2 per second. B - cat No. 43. The biopotentials

of a part of the gyrus sigmoideus post. poisoned by strychnine (1%
solution) and the respiration, a "spontaneous" convulsive potential,
are registered. B_1 - 1 second after recording B; beginning of
stimulation of the sciatic nerve of the opposite side; intensity of
stimulation 1.5 v.; frequency about 2 per second.)

In Fig. 78 it is seen that at the end of inspiration at the
poisoned point of the analysor two convulsive potentials arise. At
this time no clear changes of electrical activity are observed at the
other point of discharge (gyr. suprasylvius). At stimulation of the
sciatic nerve the rhythm of the biopotentials that arise is determined
wholly by the rhythm of the stimulation; at time of stimulation no
convulsive biopotentials arise in conformity with the respiration
and in general no changes whatsoever of electrical activity of the
cortex are observed in connection with the respiration (osc. B).
At cessation of the brief stimulation of the sciatic nerve convulsive
potentials arise in connection with alternate respiratory movement
(osc. C). At cessation of prolonged stimulation of the nerve for
a period of many seconds no convulsive potentials arise. At this time
the main electrical activity is attenuated in the part of the cortex
in question (osc. D). Then the convulsive discharges arise again at
the rhythm of the respiration, and the electrical activity of the
cortex is intensified (osc. E). Thus, it is possible to prevent the

arising of convulsive discharges by way of pure nerve influences on
the corresponding territory of the cortex.

The connection of the convulsive strychnine potentials with
the respiratory movement indicates that the "impulsation" stipulating
these strychnine potentials is connected with the respiratory act.

Concerning the irradiation of impulses from the respiratory
center through the central nervous system. Mislavskii (1885), on
the basis of experiments with cross sections and with injuries of
the medulla oblongata, by its electrical stimulation, and finally
on the basis of histological investigations, first gave an exact
description of the location of the respiratory center. He localized
it in the reticular formation (formatio reticularis) of the medulla
oblongata and expressed the consideration that it consisted of an
inspiration part and an expiration part. Sergievskii (1947) has
presented a series of physiological proofs of the existence of an
expiratory center. Pitts, Magoun and Ranson (1939) and Pitts (1940),
on the basis of experiments with point stimulation of different
points of the medulla oblongata and on the basis of histological
investigations, and Woldring and Dirken (1951), on the basis of
experiments with registration of bioelectrical potentials of the
separate neurons of the reticular formation, came to the same
conclusion. According to their data, the inspriatory center is

found in the formatio reticularis ventralis and the expiratory in
the formatio reticularis dorsalis.

It is well known that impulses from the respiratory center
are spread along the descending pathways and stipulate excitation
of the motoneurons in the cervical and thoracic parts of the spinal
cord that give origin to the diaphragmatic and intercostal nerves.

Beritov in 1915 encountered facts indicating that the
respiratory center governs rhythmical excitation of the tonic
centers of the medulla oblongata.

Kunstman and Orbeli (1921) discovered that some time after
deafferentation of a hind extremity in dog its rhythmical contractions,
synchronous with the respiration, began. On the basis of this fact,
Orbeli concluded that excitation irradiates from the respiratory
center through the central nervous system, that excitation from the
respiratory center spreads diffusely (orbeli, 1938), that this "impulsa-
tion" is insufficient for excitation of the normal centers and that
as a result of the deafferentation an elevation occurs of the
excitability of the corresponding spinal centers. All these theoretical
conclusions have been confirmed as a whole, and a number of phenomena
of the same order have been discovered.

1. With special experiments it was demonstrated that respiratory
movements of the extremities or respiratory contractions of isolated

muscles, as well as periodic intensifications of the patellar reflex

in connection with respiratory movements, are stipulated by irradiation

of impulses from the respiratory center and are not the result of

"impulsation" from lungs, respiratory tracts, or respiratory muscles

(King, Blair, and Garrey, 1931; Vinokurov, 1946).

2. On the basis of experiments with myographic registration of

respiratory contractions of isolated muscles it was observed that

these contractions can set in in conformity with either inspiration

(Vinokurov, 1945). Thus, the respiratory movement of the muscles

of the extremity can correspond to each phase of respiration, and

no selective effect of inspiratory and expiratory impulses exists

for the flexors or extensors (Sergievskii, 1950). At registration

of respiratory contractions of the muscle-antagonists it was

ascertained that the antagonists contract simultaneously (Vinokurov,

1945); nowhere was it noticed that one muscle was contracted and

the antagonist relaxed (Sergievskii, 1950). Thus, respiratory

contractions of muscles are uncoordinated acts. These facts testify

to the diffuse irradiation of impulses from the respiratory center

into the spinal cord.

3. It has been demonstrated that neurons of deafferentated

segments of the spinal cord have heightened excitability. Deafferentated

leg contracts in response to different stimulations, labyrinthine,

proprioceptive, and exteroceptive, which are subthreshold in the

sense of provocation of movement of a normal extremity (Chernevskii,

1935; Shumilina, 1945). In point experiments on cats with a hind

extremity deafferentated beforehand it was found that spinal neurons

after deafferentation (after 5-47 days) became highly sensitive to

impulses of excitation from the descending pathways (Teasdall and

Stavraky, 1953). On the other hand, it has been demonstrated that

respiratory contractions of muscles can set in also in an extremity

with normal innervation if the excitability of the spinal cord is

elevated in any way. Respiratory contractions of muscles appear

after local poisoning with strychnine of the corresponding segment

of the spinal cord (Vinokurov, 1946) and after general strychnine

poisoning, in which general twitches (Vinokurov, 1948) arise at

the rhythm of the respiration. Protracted extension of the muscles

isolated for myographic recording likewise apparently leads to

heightening of excitability of the corresponding spinal-cord centers,

as a result of which evidently respiratory movements of the muscles

in Vinokurov's experiments also arose.

Thus, from the respiratory center, at each excitation of it,

impulses irradiate into the spinal cord, showing there a diffuse

effect in the sense of rhythmical changes of degree of excitability

of its neurons and in certain conditions producing their excitation.

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Smirnov (1948) and Sergievskii (1950) came to the conclusion
that impulses of excitation from the respiratory center, irradiating
through the central nervous system, show an effect on the cerebral
cortex. The following experimental data were used for proof of this
position. 1) At cessation of respiration, caused by electrical
stimulation of the central end of the vagus nerve, chronaxy of the
motor area of the cortex is lengthened (Smirnov, 1948; Trofimov and
Raevskii, 1938; Raevskii, 1945). However, first, during stimulation
of the vagus nerve a drop of blood pressure occurs and it may be
thought that the increase of chronaxy of the neurons of the motor
area of the cortex, i.e. the reduction of their excitability, is
connected with this circumstance and not with cessation of "impulsation"
from the respiratory center. Secondly, at stimulation of the vagus
nerve bioelectrical effects arise in a certain area of the cortex
(Bailey and Bremer, 1938) and throughout the whole cortex momentary
attenuation or cessation occurs of the slow-alpha-similar fluctuations
(Zanchetti, Wang, and Moruzzi, 1952). It is possible to think that
increase of chronaxy of the neurons of the motor area of the cortex
is stipulated not by cessation of "impulsation" from the respiratory
center but by processes arising in the cortex under the influence of
impulses proceeding here at stimulation of the vagus nerve. 2) Chronaxy
of the motor area of the cortex changes depending on the phase of

respiration: it is considerably reduced at the time of inspiration
and returns to the previous level after expiration (Sergievskii,
1950). However, these facts also cannot serve as direct proof of
the irradiation of impulses from the respiratory center into the
cortex. At determination of chronaxy of the motor cortex it is
thought that its magnitude reflects the degree of excitability,
namely, ofthe cortex; thereby attention is not attracted to the
fact that at determination of chronaxy there is employed as indicator
of excitation of the pyramidal neurons of the cortex the contraction
of a corresponding group of muscles with which the pyramidal neurons
are not directly connected but (connected) through the spinal-cord
centers. It is known, furthermore, that in a number of cases
impulses proceeding along the pyramidal pathways show only subthreshold
effect on the spinal-cord neurons. This, first of all, is well known
in respect to impulses which arise in the pyramidal pathways in
connection with the alpha-waves (Adrian and Moruzzi, 1939); secondly,
a single electrical stimulation of the motor area of the cortex can
cause discharge of impulses in the pyramidal pathways, not provoking
a motor reaction since impulses proceeding into the spinal cord only
heighten the excitability of the neurons which, however, are not
discharged (Bernhard, Bohm, and Petersen, 1952). Furthermore, impulses
from the respiratory center irradiate downward into the spinal cord

and certainly stipulate a heightening of the excitability of the

spinal neurons. Smirnov (1948) concluded this, and this issues

from the fact that during certain conditions these impulses are

capable of exciting the spinal neurons. Now it is asked whether

as a result of stimulation of the vagus nerve respiration ceases

and thereby the chronaxy of the motor area of the cortex is

increased; then it is asked on what this depends: Does it depend

on cessation of "impulsation" from the respiratory center into

the cortex or on cessation of it in the spinal cord and decline

of the excitability of the spinal-cord cells, which are excited

now in response to this shock of impulses from the pyramidal path-

ways that earlier was superthreshold in this sense? From this point

of view increase of chronaxy after expiration and its abridgment at

the time of inspiration can also be connected with changes of

excitability of the spinal-cord centers during irradiation of

impulses from the respiratory center into the spinal cord, and

it is not entirely a reflection of the changes of excitability of

the cerebral cortex.

Livanov described the so-called respiratory rhythms in the

electrocorticogram of rabbits are being, in his opinion, an expression

of the cortical representation of the respiration (Livanov and

Poliakov, 1945). However, it has been demonstrated that the slow

fluctuations of potential in Livanov's recordings, as well as of
Gurevich's (1947), do not express the activity of the brain; their
arising is linked with passage of air through the nasal cavity
during breathing (Roitbak and Khechinashvili, 1952). Thus, these
data cannot be used to solve the question of irradiation of impulses
from the respiratory center into the cerebral cortex.

The following series of facts, on the one hand, serve as
additional proof of the beginning of a connection which exists
during certain conditions between the arising of convulsive
discharges and the respiration. On the other hand, on the basis
of these facts it is possible to conclude on the origin of afferent
impulses which proceed into the cortex in connection with the respiratory
act and stipulate the arising of convulsive discharges at the point of
the cortex that was poisoned.

1) At cessation of artificial respiration a more or less
prolonged pause of respiration sometimes sets in, at which time the
convulsive discharges in the poisoned part of the cortex cease; the
first convulsive discharge arises with the first respiratory movement.

2) At stimulation of the central end of sectioned vagus nerve
in a number of cases it was possible to observe parallelism in the
change of respiratory movements and convulsive discharges in the
cortex: at increase of frequency of respiration increase of frequency

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occurred of the convulsive discharges; at arrest of respiratory movements, convulsive discharges ceased. In one experiment, after bilateral section of the vagus nerves, convulsive discharges followed the slowed-down rhythm of the respiration.

3) Convulsive discharges in the cortex do not stop and do not change their character after section of the spinal cord at the boundary with the medulla oblongata (the animal finally died at artificial respiration); their rhythm corresponds to the usual rhythm of respiration of narcotized animal (Fig. 79).

4) Convulsive discharges in the cortex disappear after section of the brain at the level of the midbrain.

In a recording of Fig. 80 (osc. A) groups of convulsive potentials are visible that arise at the rhythm of respiration in the poisoned cortical point. After section of the midbrain the respiratory movements became considerably more infrequent; the convulsive potentials disappeared and were not observed for tens of minutes (osc. B). At the same time direct stimulation of the brain provoked considerable bioelectrical effects in the point of the cortex being discharged (osc. C), which indicates that the neuronic elements of the poisoned point of the cortex were capable of being excited in response to impulses proceeding to them in connection with stimulation of the surface of the cortex. Oscillogram

D shows that the cortex preserved a certain level of "spontaneous"

bioelectrical activity.

On the basis of the series of facts cited above it can be

concluded that afferent impulses proceeding into the cortex in connection

with the respiratory act do not proceed from the lungs, the air-carrying

passages, or the respiratory muscles but from the respiratory center of

the medulla oblongata. In connection with this conclusion, it is

interesting to note that Sechenov (1891) expressed the opinion that

the respiratory center in the medulla oblongata is, as it were, the

sensory surface for the cerebral cortex.

Thus, the strychnine potential arising without special discharges

in the poisoned part of the cortex is not spontaneous but stipulated by

the afferent impulses. Consequently, this convulsive discharge has

the same origin as the bioelectrical potential that arises in the

strychninized part of the analysor at peripheral stimulation, the

components of which correspond to all components of the usual primary

response, but are only extremely intensified under the effect of the

poison.

Thus, the initial positive potential of "spontaneous" convulsive

discharge expresses local excitation of the elements of layers III

and IV that arise under the effect of impulses from the respiratory

center. Its lesser amplitude, at comparison with the positive phase

of the strychnine effect provoked by stimulation of a nerve, testifies

to the fact that afferent "impulsation" from the respiratory center

at each given point of the cortex is minimal and that, with normal

excitability of the cortex, impulses from the respiratory center do

not provoke appreciable direct biopotentials that would be isolated

on a background of "spontaneous" electrical activity, whereas the

weakest peripheral stimulations, for example stimulation of several

skin receptors, provokes in the cortex a characteristic positive

potential. There is not a single case in which bioelectrical

potentials are disclosed only under the effect of strychnine. For

instance, stimulation of the optic nerve causes no visible bioelectrical

effects in the part of the gyrus ectosylvius around the suprasylvius

fissure; strychnine effects arise here after strychnine poisoning

of this part in response to stimulation of the optic nerve (Clare and

Bishop, 1954). The positive potential of "spontaneous" convulsive

discharge usually lasts a longer time than the corresponding component

of the provoked strychnine effect (Figs. 77 and 78). This can be

explained, first, by the fact that from the respiratory center flows

a synchronous shock of impulses considerably less as compared to

those proceeding into the cortex at peripheral stimulations. Secondly,

we have seen that the length of the positive phase can be altered,

depending on how quickly the negative phase arises after it. It can

be thought that a comparatively great interval of time should elapse
before local excitation of such intensity spreads to the neurons to
which a small number of the asynchronous impulses from the respiratory
center proceeds, at which they begin to discharge impulses of excitation.
Apparently from the respiratory center in the cortex, in connection
with excitation by the inspiratory and expiratory part of it, a long
series of impulses is directed, starting approximately 0.1 sec. before
the beginning of inspiration or expiration and lasting for almost as
long a time as the inspiration or expiration lasts. This can be
concluded on the basis of data obtained during a study of the electrical
activity of the separate cells of the respiratory center (Dirken and
Woldring, 1951). That one strychnine potential, or a group of 2-3
potentials separated by pauses of 0.2 sec. and more, arises in the
poisoned part of the cortex in response to a whole series of afferent
impulses is fully explicable, since a like reaction arises in a
strychninized point of the skin analysor at comparatively frequent
stimulation of the sciatic nerve.

The negative phase of the strychnine discharge, which as
McCulloch showed (1949a) is removed by thermocoagulation of the
upper layers of the cortex, is an expression of the activation of
the top dendrites of layers I and II under the effect of a great
number of impulses that reaches them through the ascending axons

of the intermediate neurons and through the ascending collaterals
of the pyramidal neurons. If, first, it is considered that at
the strychninized point of the cortex pyramid neurons of all layers
and numerous neurons with short axons are stimulated, then, secondly,
that at strychnine poisoning each neuron is stimulated by impulses
at a rhythm of 500-900 per second, and, thirdly, that under the
effect of the strychnine the elementary local potential is intensified,
if these circumstances are considered, then the gigantic amplitude of
the negative phase of the strychnine potential becomes apparent.

The positive fluctuation arising after the negative is probably
the expression of a preeminent activation of the neurons of the deep
layers. This potential is associated with discharge of impulses into
the white matter (McCulloch, 1949a; Baumgarten and coworkers, 1954).

(Legend to Fig. 79, textp. 202: Convulsive discharges in the
cortex before and after section of the spinal cord at the level of
the medulla oblongata. Cat No. 51, Feb. 14, 1952. Potentials are
discharged from a part of the gyrus suprasylvius poisoned with
strychnine (1% solution). Recordings A and B were made prior to
section of the cord. A - during artificial respiration. B (lower
left) - during natural respiration. C (lower right) - after section
of the spinal cord; artificial respiration.)

(Legend to Fig. 80, textp. 203: Effect of section of the

midbrain on convulsive discharges. Upper curves - bioelectrical
potentials from a part of the gyrus suprasylvius poisoned by 1%
strychnine; lower curves - respiration, rise upward - expiration.
A - convulsive potentials at the rhythm of the respiration. B -
cessation of convulsive discharges after section of the midbrain.
C - effect of direct electrical stimulation of the cortex;
stimulating electrodes 2 mm. from part poisoned; frequency of
stimulation 1 per second, intensity 15 volts. D - electrical
activity of poisoned part at greater intensification (Roitbak,
1954d).)

McCulloch (1949), on the basis of other facts and considera-
tions, came to an analogous conclusion on the origin of the three
phases of the strychnine potential.

The local character of the convulsive discharges is explained
by the development of a process of inhibition; evidently the negative
potential expressing local excitation of the top dendrites, not only
the first to be excited but also the adjacent ones of the unexcited
neurons, is the reason for oppression of the excitability of the
latter (see above). The negative potential should be the larger in
amplitude the more intense the excitation of the neurons, the axons
of which ascend into layer I; thus, intensive excitation of poisoned
neurons stipulates intensive inhibition of neurons adjacent to them,

which also impedes excitation spread from the strychninized point
along the cortex. So is it possible to explain this phenomenon
proceeding from the dendritic hypothesis of inhibition. (However,
if several points of the cortex lying in a line are poisoned with
strychnine, then the convulsive discharge provoked in one of them
spreads to the others if the distance between the poisoned points
does not exceed 6 mm. This phenomenon also occurs in isolated strip
of cortex, i.e. excitation spread from one poisoned point to another
can proceed without participation of subcortical development.
Excitation-spread rate in these conditions is 0.15-0.25 m. per
second (Cobb and coworkers, 1955). Thus, convulsive discharges are
limited by the poisoned point of the cortex in case of normal
excitability of the surrounding points of the cortex; if their
excitability is elevated, then the process of inhibition cannot
"check" the spread of the excitation process to new complexes of
neurons.)

Purely negative "spontaneous" strychnine potentials, i.e.
convulsive potentials without an initial positive phase, have been
described by some authors. In the course of the present investigation
this also was observed. The arising of such sort of potentials can
be explained the same as the arising of purely negative potentials
in response to peripheral stimulations: the very first afferent

impulses provoke a most intense discharge of the neurons of layer IV

and the arising of a purely negative potential, since cohesions of

a small number of initially excited neurons are not intercepted from

the surface of the cortex. On the other hand, it is theoretically

possible to assume that the center of poisoning with the usual +-+

potentials must be surrounded by an aureole of purely negative

potentials (see textpage 189) and if the discharge electrode is

located in this territory, then negative potentials are registered

without a preceding positive phase. In the initial phase of strychniniza-

tion of the cortex purely negative strychnine potentials can arise.

They are not associated with spread of excitation impulses from the

poisoned part to other territories of the cortex and into the subcortex,

i.e. these negative potentials are not attended by excitation and by

discharge of the pyramidal neurons. If the poison continues to lie in

place, then the usual three-phase (+-+) strychnine potentials begin to

arise (McCulloch, 1949a). It is possible to think that in the beginning

stage of local poisoning, so long as the poison does not penetrate into

the middle and deep layers, increase of excitability of the intermediate

neurons of layer II occurs. At being excited, they stipulate the

arising of a purely negative potential, the arising of which thus is

the same as of the supplementary negative potential in effect of electrical

stimulation of the cortex (compare Fig. 23).

As already said, convulsive discharges arise at the rhythm of
the respiration in any part of the cortex after its poisoning with
strychnine. At poisoning of several parts, in each such part convulsive
bioelectrical discharges set in linked in one way or another with the
rhythm of the respiration, but entirely independent of the convulsive
activity of other poisoned cortical parts. This circumstance has
been noted by other investigators too (Beritov and Gedevanishvili,
1945). Thus, at poisoning of two points of the cortex, for instance
of the gyrus sigmoideus and gyrus suprasylvius, and simultaneous
registration of convulsive discharges from these two points it is
possible to observe that, arising in connection with one or another
phase of respiration, these discharges rarely arise synchronously,
the effect at point 1 can precede the effects at point 2; the effect
at point 1 can arise without the effect at point 2 arising at this
time; at point 1 convulsive discharges can arise in conformity with
each inspiration and at point 2 in conformity with each expiration;
at point 1 single spasmodic discharges can arise, and at point 2
groups of them, etc. After separation of the poisoned points of the
cortex by section of the cortex between them, convulsive discharges
continue to arise at the rhythm of the respiration at both points of
the cortex, and their character is not altered.

Nevertheless, at poisoning of two points of one convolution

(for instance, of the suprasylvius) convulsive potentials can arise

entirely simultaneously at both points both before and after their

separation (Fig. 81, textp. 206: Cat No. 48. Dec. 11, 1951.

Discharge electrodes E_1 and E_2 are placed on the surface of the

gyrus suprasylvius; distance between them 12 mm. Local poisoning

of the cortex with strychnine (1% solution) was made simultaneously

under E_1 and E_2. The potentials are discharged from point E_1 (upper

curves) and from point E_2 (lower curves). A - convulsive potentials

arising at the rhythm of the respiration after poisoning under E_1

and E_2. After this, deep section was made of the gyrus suprasylvius

between E_1 and E_2. B - convulsive potentials arising at the rhythm

of the respiration after section. C - the same at greater intensification.).

It should be confirmed that stimulation of auditory, optic,

vestibular, skin, and other receptors leads to the arising of the

direct bioelectrical effects already described in limited areas of the

cortex, in the nuclei of the corresponding analysors, and does not

arouse them in other territories of the cortex, even at local strychnine

poisoning of the latter. Impulses too from the respiratory center, being

afferent for the cortex, can at strychnine poisoning provoke effects

characteristic to afferent stimulation in any part of the cortex. Thus,

impulses from the respiratory center show a widespread influence on the

cortex.

The part of the cerebral cortex poisoned by strychnine is the focus of heightened excitability, capable of being excited under the effect of the weakest "impulsations" reaching it. We have seen that such a focus is excited under the action of impulses from the respiratory center. If it is created in a corresponding part of the skin projection territory of the cortex, then its excitation occurs at stimulation of the sciatic nerve. At a stimulation frequency of 2-15 per second strychnine potentials arise at the rhythm of the stimulation. Impulses from the respiratory center do not now provoke excitation at this focus and are not reflected on the effects of stimulation of the sciatic nerve. Thus, the excitation rhythm of the strychninized center of the cortex is now determined not by the rhythm of excitation of the respiratory center but by the rhythm of stimulation of the sciatic nerve. This point of the cortex is capable of being excited under the effect of each of these two stimulations separately, but at their simultaneous action it "is subordinated" to the more powerful of them. Apparently in the pauses between shocks of stimulation of the sciatic nerve impulses from the respiratory center are incapable of provoking excitation of the poisoned neurons because of a decline in them of excitability after intensive excitation.

A like phenomenon is observed at direct electrical stimulation of the cortex not far from the poisoned portion. For instance, in one

experiment two points of the cortex were poisoned, one in the region
of the gyrus sigmoideus, the other in the region of the gyrus
suprasylvius. At both points strychnine potentials arose at the
rhythm of the respiration. At electrical stimulation of the cortex
in the region of the gyrus sigmoideus at the rhythm of 10 per second
strychnine discharges here were sharply increased in frequency,
their rhythm was determined by the rhythm of stimulation of the
brain, and each discharge arose in response to a shock of stimulation.
The rhythm and character of the discharges in the parietal region
were not altered, and they continued to set in at the rhythm of the
respiration.

We have already run into a similar phenomenon during analysis
of bioelectrical potentials in the foci of heightened excitability,
created by tetanization of the surface of the cortex: if at the time
of the aftereffect electrical stimulation is applied to any point
at all of the cortex, then "spontaneous" rhythm of the potentials
of the aftereffect is changed by the rhythm of the stimulation applied
even in case it is more infrequent than the rhythm of the "spontaneous"
biopotentials of the aftereffect (Figs. 50 and 51). Thus, the focus
of heightened excitability reacts to the most intensive of all
stimulations that fall on it at a given moment and cannot react to
the rest. If this stimulation is removed, it begins to react to other

stimulation which proves more intense than the rest.

(Legend to Fig. 82, textp. 209: Periodical changes of bio-
electrical activity in cerebral cortex of cat and of alpha-rhythm
in man in connection with respiration. Recordings A and B from two
different cats under nembutal narcosis. In both cases potentials
are registered from the gyrus sigmoideus post. (upper curves) and
from the gyrus suprasylvius (lower curves) and the respiratory
movements of the chest are registered. Recordings C and D in
the individual tested when the eyes were closed at cessation of
prolonged work on the bicycle dynamometer. There are registered:
respiratory movements of the chest, the electroencephalogram, and
the electrocardiogram (Roitbak, 1953c). Indications for A: 0.5
sec. and 0.3 milliv.; for D: 20 millisec. and 40 microv.)

The influence of the respiratory center on the cerebral
cortex can be disclosed oscillographically in animals without
poisoning the cortex with strychnine. At a certain depth of nembutal
narcosis, from the cerebral cortex the rhythm, described by many
authors, of slow fluctuations with a frequency of about 10 per second
is discharged. Fluctuations of this rhythm set in by series separated
by pauses or, from time to time, an increase of the amplitude of these
fluctuations occurs. At attentive observation it is possible to
ascertain that intensification of slow fluctuations of the bioelectrical

potential often occurs in the electrocorticogram at the rhythm of
the respiration. Hence, it is possible to conclude that in animal
during normal and even reduced excitability of the cortex the effect
of afferent "impulsation" from the respiratory center is expressed
in a periodical intensification of the slow cortical biopotentials
(Fig. 82, A and B).

As known, the alpha-waves of man set in serially; in each
series their amplitude is progressively increased and then progressively
reduced. The length of these series ("spindles") varies from 0.5 to
3 sec. (Beritov, Bakuradze, and Dzidzishvili, 1943; Chugunov, 1950).
The reasons of this phenomenon of the so-called "slow periodicity"
remain unknown. In connection with this, the following observation
offers a certain interest.

In certain conditions it is possible to observe a regular
connection of periodic intensification of the alpha-waves with the
respiration. This is observed, for example, at intensified respiration
at the time of and at cessation of prolonged intensive work (revolving
the pedals of a bicycle dynamometer). In Fig. 82, C and D, it is
seen that the frequency of the series of alpha-waves corresponds to
the frequency of the respiration. (Rhythmical heart activity finds
no reflection in the electroencephalogram).

It is possible to assume that periodic changes of the alpha-

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rhythm, synchronous with the respiration, are stipulated by "impulsation"
from the respiratory center (Roitbak, 1953c). (In Stroup and Darrow's
article (1953) it is also noted that spindles of alpha-waves in man
can be linked with intensified respiratory movements and that this
probably occurs because of the influence of the respiratory center in
the cortex.)

Moruzzi and coworkers (1950) and Chang (1952) have described
the phenomenon of periodical abatement and intensification of cortical
potentials arising with different methods of excitation of the cortex:
at stimulation of the sciatic nerve (primary responses in the skin
projection territory), at stimulation of the corpus geniculatum
laterale (primary responses in the optical projection territory),
and at stimulation of the pyramidal pathways (antidromic effects
in the motor area of the cortex). All these potentials, at uninterrupted
stimulation at an infrequent rhythm periodically intensify and attenuate.
The nature of this phenomenon was not clarified. According to Chang,
this is the development of a certain general property of the central
nervous system. However, it was ascertained that intensification of
the potentials provoked coincides with the arising of an alternate
"spindle" of alpha-similar waves. In connection with what was stated
earlier, it is possible to assume that periodic changes of the answering
bioelectrical reactions of the cortex can be stipulated by periodic
changes of its excitability under the influence of impulses from the

respiratory center.

The activity of the subcortical nerve centers is governed by the cerebral cortex, because of which this activity of the subcortical centers proves to be in obligatory conformity with the vital state of the animal (Pavlov, 1930); particularly well known to us is the governing role of the cerebral cortex in regard to respiration. (See Bykov, 1947; Marshak, 1949; Sergievskii, 1953; Dedabrishvili, 1953, 1954.)

On the other hand, there are numerous data indicating that subcortical formations show an influence on the cortex (see below). Pavlov, on the basis of a study of conditioned-reflex activity of the cerebral cortex, came to the conclusion that the subcortex shows a continuous influence on the functional state of the cortical elements. "...the countereffect of the subcortical centers on the cerebrum is by no means less substantial than (that of) the hemispheres on them. The active state of the cerebrum is constantly sustained, thanks to stimuli proceeding from the subcortical centers" (Pavlov, 1930); "the subcortex can... elevate the excitability of the cortical cell" (Pavlovskie sredy (Pavlov's Media), 1: 147).

(Opinion) being based on the facts cited in the present communication, it is possible to assume that in regard to constant maintenance of the active state of the cortex, of its tone, and of

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the specific degree of its excitability, an outstanding role belongs
to the respiratory center of the medulla oblongata.

Thus, the respiratory center, whose activity is regulated by
the cerebral cortex, shows a spread effect on the cortex, which is
expressed in the rhythmical changes of its excitability and, in certain
conditions, in the excitation of its neuronic elements.

Issuing from this, a theoretical clarification can be given
to many facts from sport physiology, for example to the fact of the
change in magnitude of muscle strain depending on the phase of
respiration (Farfel' and Freidberg, 1948). From this point of view,
the physiological significance of intensification of respiration,
which occurs in mammals in response to all forms of external stimulations,
even when the latter are so weak that they do not cause oriented
movements, becomes intelligible (Dedabrishvili, 1953, 1954): apparently
the intensified activity of the respiratory center leads to a general
elevation of cortical excitability. Finally, the fact that several
intensified respiratory movements can in certain patients with epilepsy
lead to the arising of convulsive electrical activity can be explained
not by the change in chemical composition of the blood resulting from
hyperventilation but by purely nerve influences on the cerebral cortex
from the respiratory center. However, all these hypotheses are necessary,
finally, in special arguments.

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In what way is a diffuse, spread effect of the respiratory center realized in the cortex?

Reticular formation of the brain stem. The reticular formation (formatio reticularis) is a peculiar structure stretching through the whole brain stem. This formation begins as far back as the spinal cord, then, spreading, fills the greater part of the medulla oblongata, crosses into the midbrain, and ends at the anterior poles of the optic papillae of the midbrain. It occupies everywhere a central position and is surrounded on all sides by nerve nuclei and by conductive courses. The reticular formation, a heterogeneous mass particularly in the reticular formation of the midbrain (thalamus ventralis), is an isolated series of nuclei; in the medulla oblongata, in certain regions of this formation, are located the respiratory center, the vasculomotor center, etc. The reticular formation of the higher vertebrates has been insufficiently studied histologically, but the diffuse, undifferentiated character constituting its nerve mass is conspicuous (see Beritov, 1948).

Beritov first pointed out the important physiological importance of the reticular formation of the stem part of the brain, undertook a systematic physiological study of the reticular formation, and found that its main function consists of general inhibition and general facilitating effects (Beritov, 1937c; 1948).

The mechanism of the general inhibition and facilitating effect

of the reticular formation of the brain stem has been presented in
the following way. Beritov considered this point of view, that the
central nervous system is constructed according to the neuronal-
neuropil type. A neuropil (in the sense of Herrick) is a comparatively
undifferentiated nerve mass filling, in the central nervous system,
all space free of differentiated centers and courses. The reticular
formation consists mainly of neuropils in this sense. Furthermore,
it was admitted that the neuropil is a substrate for inhibition,
whereas the differentiated nerve centers and pathways serve for
conduction of excitation. At activation of the neuropil, which
proceeds simultaneously with the excitation of some differentiated
reflex centers, slow fluctuations of bioelectrical potential arise
in it and the neuropilic current penetrates diffusely into the nerve
pathways and centers lying nearby, stipulating their electrotonization
or their change of excitability (Beritov, 1937, 1941, 1948). However,
first, the term itself of "neuropil" failed and led to many misunder-
standings. Secondly, it is hardly correct to connect the process of
inhibition with the activity of a special undifferentiated nerve
substrate. Thirdly, as has been pointed out, the possibility of
diffuse and territorially spread effect of bioelectrical potentials
is not confirmed experimentally. Finally, from this point of view
it is extremely difficult, if not impossible generally, to clarify
in a purely physical way in what manner the anelectrotonic effect

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of neuropilic current is realized on the neuronic elements. At
the present time the mechanism of the process of inhibition is
considered differently (see Beritov and Roithak, 1955).

Issuing from the hypothesis of Beritov concerning the fact
that the reticular formation of the stem of the brain is capable
of showing on the central nervous system a general facilitating
effect, it might be possible to assume that the diffuse irradiation
of excitation from the respiratory center on the spinal cord, on
the one hand, and on the cerebral cortex, on the other hand, proceeds
by means of the reticular formation of the stem of the brain, in
the bulbar part of which is found the respiratory center itself.
As will be shown below, electrophysiological data on the working
principle of this formation support the same hypothesis.

At electrical stimulation of any part of the reticular
formation, responding bioelectrical potentials are registered over
all other parts of it; for instance. at stimulation of the formatio
reticularis of the medulla oblongata, they are discharged from the
formatio reticularis of the midbrain, sub- and hypothalamus, and
thalamus. Contrariwise, at stimulation of the formatio reticularis
of the midbrain potentials are discharged from the formatio reticularis
of the medulla longata. At the same time the adjacent surrounding
nerve formations, for instance the well-known differentiated nuclei

of the thalamus, give no bioelectrical reaction (Starzl, Taylor,
and Magoun, 1951a). Thus, at stimulation of any point of the
reticular formation the whole reticular formation of the stem of
the brain comes into an active state. However, it does not react
as a single formation: in response to a given stimulation these
or other nuclei of this formation are activated to a more or less
degree (Hanbery and Jasper, 1953; Hanbery and coworkers, 1954).

At stimulation of any part of the reticular formation,
from medulla oblongata to anterior pole of the thalamus, response
bioelectrical potentials are registered in the cerebral cortex
(Starzl and coworkers, 1951a). However, in high distinction from
the strict local character of cortical potentials arising at
stimulation of the different receptors and organs of sense, at
stimulation of the reticular formation response potentials arise
over the whole cortex, in any case they can be discharged from any
point of the dorso-lateral surface of the cerebrum (Fig. 83, textpage
212: "Diffuse" activation of the cerebral cortex at stimulation of
the anterior ventral nucleus of the thalamus. Depiction of the
cerebral cortex of cat with marked portions,/from which bioelectrical
potentials were registered at stimulation of the nucl. ventralis
anterior by bipolar electrodes with interpolar distance of 1 mm.
The small black circules in the lower illustration mark the places

where the stimulating electrodes were placed in the thalamus

(Hanbery and Jasper, 1953).).

All sensory nerve pathways bearing afferent impulses from
periphery to cortex terminate in certain differentiated sensory
nuclei of the dorsal thalamus, for instance the optic in the corpus
geniculatum laterale, the auditory in the corpus geniculatum mediale,
and the fibers of the medial plexus bearing impulses from the skin
receptors terminate in the ventro-postero-lateral nucleus (nucl.
ventro-postero-lateralis). At punctual electrical stimulation of
these nuclei bioelectrical potentials arise in certain limited
regions of the cortex, in the so-called projection territories of
the cortex, i.e., at stimulation of a given sensory thalamic nucleus,
response potentials arise in the same region of the cortex as at a
corresponding peripheral stimulation. The character of the cortical
potentials is the same, but the latent period of their arising is
less (Chang, 1952). At punctual stimulation there is discovered a
whole further series of nuclei projecting to certain limited parts of
the cortex, i.e. of nuclei from which typical primary responses
with small latent period are provoked, for instance the nucl. ventralis
lateralis is linked with the motor region of the cortex and the nucl.
lateralis post. and pulvinar with the gyrus suprasylvius (Fig. 84,
textp. 214: Scheme of local diffuse effect from thalamus to cortex.

GL - corpus geniculatum laterale; this is a nucleus directly
connected with the optic projection region of the cortex. GM -
corpus geniculatum mediale; this is a nucleus directly connected
with the auditory projection region of the cortex. LP - a nucleus
directly connected with the gyrus suprasylvius. VPL - a nucleus
directly connected with the cutaneous projection territory of the
cortex. VL - a nucleus directly connected with the region of
the motor analysor. The connections of these nuclei with the
cortex are indicated by unbroken lines. The dotted lines depict
diffuse connections of the central nuclei of the thalamus with the
cortex. The scheme was composed on the basis of known physiological
and neurological data.). Precise and limited projection of these
thalamic nuclei in the cortex is demonstrated not only by electro-
physiological but also by histological data. After removal of a
certain limited cortical field (corresponding to a certain projection
region of the cortex), retrograde degeneration of the cells of a
certain thalamic nucleus occurs (Rose and Woolsey, 1949).

In contrast to the strictly local bioelectrical reactions
arising in the cortex at stimulation of the above-mentioned thalamic
nuclei, at stimulation of the central zone of the thalamus by a
seized reticular formation bioelectrical response reactions arise
over the whole cortex. This fact was established by Morison and

Dempsey (1942) and then confirmed by many researchers (see Jasper

and coworkers, 1955).

Activation of the whole cortex occurs at stimulation of the

following thalamic nuclei: centrum medianum, ventralis med.,

intralaminaris, ventralis ant., and reticularis. Effects arising

in the cortex at single stimulations of the so-called "diffuse

thalamic system" is distinguished by a greater latent period, 15-

60 milliseconds (Jasper, 1949). However, at stimulation of the

foremost parts of this formation (nucl. ventralis ant., nucl.

reticularis) effects set in over the whole cortex with a short

latent period (Hanbery and coworkers, 1953, 1954). At stimulation

of a more medially located part of the reticular formation of

the thalamus, the anterior cortical fields are more highly activated.

At stimulation of the lateral part of this formation the posterior

cortical fields are more highly activated (Hanbery and coworkers,

1954).

It was possible to suppose that the effect of the reticular

formation on the cortex is realized by means of differentiated

thalamic projection nuclei. However, this effect is preserved

after destruction of all specific projection nuclei of the thalamus

(Hanbery and Jasper, 1953). This effect is not realized through

the strial system (Hanbery and coworkers, 1954). Thus, it is

possible to consider it established that the reticular formation

of the stem of the brain shows a widespread effect on the cortex through

its anterior part, through a "diffuse, thalamo-cortical projection

system", i.e. that the reticular system of the brain stem possesses

definite nerve pathways into the cortex independent of the well-known

pathways of specific projection systems (see Fig. 84).

The spread of excitation at stimulation of the reticular

formation occurs within the reticular formation proper through

neuronic circuits of varied degree of complexity, by which is also

explained the long latent period of the cortical reactions at

stimulation of the reticular formation. Stimulation of the reticular

formation of the medulla oblongata continues to provoke diffuse

activation of the cortex after intersection of all ascending afferent

systems, frequently of the medial and lateral plexus. It has been

demonstrated also that changes of electrical activity of the cortex,

at stimulation of the reticular formation of the medulla oblongata,

are not connected with stimulation of the descending cortical pathways,

ie. with antidromic spread of excitation through the pyramidal and

extrapyramidal pathways (Moruzzi and Magoun, 1949). Stimulation of

the reticular formation stops provoking bioelectrical reactions of

the cortex after intersection of the reticular formation in front

of the place of stimulation (Moruzzi and Magoun, 1949).

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The terminal portion of the reticular formation where reticular
impulses come is, according to latest data, the nucl. reticularis
thalami; on this nucleus lie the end neurons of the reticular system,
the axons of which proceed through the inner capsule to all parts of
the cortex (Hanbery and coworkers, 1954). Apparently these fibers
are Lorente de No's "nonspecific afferents" (see Morison and Dempsey,
1942). (The short latent period of the bioelectrical effects at
stimulation of the anterior parts of the reticular formation of the
thalamus is explained by the fact that stimulation occurs thereby
of fibers proceeding from here to the cortex (Hanbery and Jasper, 1953).)
It is characteristic that at removal of any projection region of the
cortex retrograde degeneration arises only in a small part of the
nucl. reticularis. Degeneration of cells in the whole reticular nucleus
sets in only as a result of extirpation of the entire cortex (Rose
and Woolsey, 1949; Hanbery and coworkers, 1954).

There are indications that, besides this pathway, by which the
reticular effects to the cortex are realized, there is a further pathway
into the cortex through the sub- and hypothalamus (Starzl and coworkers,
1951a).

Stimulation of the reticular formation provokes an effect in a
given part of the cortex even after its isolation from the rest of the
cortex (Dempsey and Morison, 1942; Jasper and coworkers, 1955), or after

extirpation of the rest of the cortex (Hanbery and coworkers, 1954).

Consequently, it is impossible to explain the diffuse bioelectrical

reaction of the cortex at stimulation of the reticular formation by

the fact that impulses from the formatio reticularis reach any limited

part of the cortex and right from there, by means of intracortical

connections, excitation spreads through the entire cortex.

It is proposed that thalamic reticular formations of the left

and right sides are connected with one another by means of the massa

intermedia or by means of subthalamic formations; because of this,

at unilateral stimulation of the reticular formation, the cortex of

both cerebral hemispheres is activated, as has been said (Hanbery

and coworkers, 1954).

Thus, the cerebral cortex receives excitation impulses, first,

from differentiated specific nuclei of the thalamus excited in isolated

fashion at corresponding peripheral stimulations that transmit excitation

into strictly specific limited projection territories of the cortex and,

secondly, from the so-called "diffuse thalamo-cortical system" excited

at stimulation of any point of the reticular formation of the brain stem

and transmitting excitation into all parts of the cortex.

It is possible with great probability to think that the effect

of the respiratory center on the cerebral cortex is realized thanks

to the spread of excitation through the whole reticular formation of

the brain stem and then through a "diffuse thalamo-cortical system"
to the whole territory of the cerebral cortex.

Characteristic bioelectrical reactions of the cortex at
stimulation of the reticular formation. As said, in response to
single stimulations of any point of the reticular formation from
all points of the cortex it is possible to register a responding
bioelectrical reaction. The potentials arising at stimulation of
the different parts of the reticular formation of the thalamus
have been studied in detail (Dempsey and Morison, 1942; Jasper,
1949; Verzeano, Lindsley, and Magoun, 1953). The characteristic
reaction in response to a shock of stimulation is a two-phase
potential arising with a long latent period. The first phase,
the positive, usually is weakly expressed and proceeds without
pause into the negative potential of greater amplitude. At sinking
the discharge electrode more deeply into the cortex the "inversion"
of the potential occurs at a depth of 1-2 mm., i.e. the initial
positive fluctuation expresses regional excitation of the neurons
of the middle and deep layers of the cortex for the most part, and
the negative fluctuation expresses the regional excitation of the
elements of the surface layers of the cortex. Under the influence
of strychnine both phases are intensified. At narcosis and at
asphyxia the negative phase is the first to disappear (Verzeano
and coworkers, 1953).

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At stimulation of a given point of the reticular formation
of the thalamus, responding potentials arise in the different
territories of the cortex with a different latent period, for instance
at stimulation of the nucl. centr. med. the latent period of the
potential in the frontal region of the cortex equals 20 milliseconds,
in the parietal region 40 milliseconds (Jasper, 1949).

After the responding biopotential considered, a series of slow
potentials can arise at a rhythm of 8-12 per second (Jasper, 1949).

At repeated stimulations at a rhythm approximating the rhythm
of the alpha-waves and in response to the first 5-6 shocks of
stimulation, a progressive increase of bioelectrical effects is
observed, then they weaken with the same gradualness and dwindle
almost to nothing; however, at unceasing stimulation, they arise
again after a certain interval of time, gradually increasing to
reach a maximum, and then gradually dwindle to nothing, etc. A
similar phenomenon was recorded by Narikashvili (1953), also on
non-narcotized animals with electrodes reanimated in the subcortex
and cortex, at stimulation of the central parts of the stem of the
brain. The periods of time between the "spindles" so arising are
the same as the periods of time between the "spontaneous" series
of alpha-waves.

The above-described characteristics of the cortical potentials

arising at direct stimulation of the reticular formation fully
correspond to the site that the respiratory center affects in the
cerebral cortex through the reticular formation: 1) strychnine
potentials arising at the rhythm of the respiration have a weakly
expressed initial positive fluctuation; 2) they do not arise in
different regions of the cortex simultaneously; 3) after the
strychnine potential that has arisen under the effect of impulses
from the respiratory center, a group of alpha-similar waves some-
times follows (Fig. 81); 4) finally, periodic intensification
and abatement of the alpha-waves can arise at the rhythm of the respiration.

According to Beritov's data, the reticular formation of the stem
of the brain should come into an active state of each peripheral
stimulation: together with excitation of certain differentiated nerve
pathways, nuclei, and centers, excitation occurs of the undifferentiated
mass of the reticular formation (1937, 1948, etc.). This aspect can
at the present time be considered demonstrated, since it has been
established by histological and electrophysiological methods of
investigation that from all ascending sensory pathways (medial plexus,
lateral plexus), through the whole course of their excitation to the
corresponding thalamic projection nuclei, collaterals go out into the
reticular formation of the stem of the brain. At stimulation of the
sciatic nerve and at sound stimulations, activation occurs of the

whole reticular formation from the medulla oblongata to the anterior

pole of the thalamus (Starzl and coworkers, 1951b; see also Bremer

and Terzuolo, 1952). The same was established in regard to the optic

system and afferent system of the n. splanchnicus (see in Verzeano

and coworkers, 1953). In most recent investigations it has been

shown that the separate cells of the reticular formation of the

medulla oblongata are excited at stimulation of the sciatic nerve and

of the trifacial nerve (Baumgarten and coworkers, 1954).

On the other hand, it has been found that the reticular formation

of the midbrain is activated at electrical stimulation of the gyrus

ectosylvius and gyrus suprasylvius (Bremer and Terzuolo, 1952), as

well as at stimulation of the cortex in the region of the cutaneous,

motor, and optical analysors (Bremer and Terzuolo, 1954). At

strychninization and at electrical stimulation of the motor territory

of the cortex excitation or inhibition occurs of the separate cells

of the reticular formation of the medulla oblongata (Baumgarten and

coworkers, 1954). Thus, the reticular formation of the stem of the

brain is also activated by collaterals from pathways descending from

the cortex, particularly from the pyramidal pathways. (It has been

clarified, furthermore, that the reticular formation of the medulla

oblongata receives afferent fibers from the cerebellum (Baumgarten

and coworkers, 1954), and the reticular formation of the thalamus

receives fibers from the caudate nucleus (Shimamoto and Verzeano, 1954).)

In response to a single stimulation of the sciatic nerve at relatively deep barbituric narcosis a peculiar generalized bioelectrical reaction of the cortex is disclosed: with a latent period of 40-80 milliseconds, a slow potential arises 40-80 milliseconds in length; in the skin projection territory of the cortex a primary response, setting in with a latent period of 10 milliseconds precedes this potential. A second shock of stimulation gives an effect at an interval of more than 0.5 millisec., but for complete restoration of the magnitude of the effect an interval of 1 sec. and more is required. At a stimulation frequency of 5 per sec., the reaction described (the "secondary effect") arises only in response to the first shock of stimulation. This reaction arises over the whole cortex. The reaction is entirely identical in the hemisphere in question whether the sciatic nerve of the opposite or of the correspond-ing side is stimulated, although the primary response is weaker or lacking at ipsilateral stimulation. The latent period of the "secondary effect" is extremely prolonged under the effect of ether, which generally quickly removes it. The secondary effect is similar in character to the separate "spontaneous" slow potentials arising from time to time (Derbyshire and coworkers, 1936; Forbes and Morison, 1939).

At examination of the galvanometric recordings of Pravdich-Neminskii (1925) it is possible to conclude that in his experiments he actually dealt with the generalized "secondary response" of the cortex considered above, having discovered, recorded, and described it long before the American investigators (see textpage 135). Evidently the "secondary" bioelectrical reaction of the cortex is realized by means of the reticular formation of the stem of the brain.

It is well known that under the influence of peripheral stimulations in normal animals found in a sleeping state or simply in a state of rest and in lightly narcotized animals a generalized reaction arises consisting of this, that relatively slow and infrequent fluctuations of biopotential give way to relatively quick and frequent ones, i.e. a reaction arises analogous to the reaction of depression of the alpha-rhythm in man (Gershuni and Tonkikh, 1946, 1949; Tskipuridze, 1950a; Narikashvili, 1950; Jasper and coworkers, 1955, et al.). In the sense of the provocation of this generalized reaction the most effective are the pain stimulations, less effective are the propriocep-tive stimulations, still less effective are the sound stimulations, and optical stimulations have the weakest of all influence (Bernhaut, Gellhorn, and Rasmussen, 1953). In certain conditions peripheral stimula-tion can provoke the paradoxical reaction of the intensification or of the appearance of the alpha-rhythm. This was observed in people, for

instance, in a certain stage of sleep and with schizophrenia

(Subbotnik and Shpil'berg, 1947; Roitbak and Savaneli, 1952, 1953).

In animals this was observed in a certain stage of barbituric

narcosis (Adrian, 1941).

Furthermore, it was found that stimulation of the reticular

formation of the stem of the brain at a rhythm of 50-300 per second

provokes the same reaction in the cortex (Moruzzi and Magoun, 1949;

Bremer and Terzuolo, 1954). Hence, Moruzzi and Magoun concluded that

the reaction of awakening under the effect of external stimulations

proceeds through the medium of the reticular formation of the stem of

the brain, called the "activating system" of the brain. This fact

was also used for such a conclusion, that stimulation of any projection

nucleus of the thalamus at a rhythm of 50-300 per second causes the

appearance of quick potentials only in the corresponding projection

territory, i.e. it does not provoke generalized reaction of the cortex

(Starzl and coworkers, 1951a). Accordingly, the reaction of falling

asleep and sleep (natural and narcotic) were to be explained by the

cessation or abatement of the "impulsation" from the reticular formation

of the stem of the brain into the cortex. In favor of such a conclusion

this fact was used, that isolated stimulation of the fore part of the

reticular formation of the brain stem causes sleep in an animal and

the picture of electrical activity of the cortex characteristic to

sleep. No such phenomena were observed at isolated section of the main ascending afferent pathways (Lindsley and coworkers, 1950).

However, Pavlov showed incontestably that sleep can arise initially in the cerebral cortex under the effect of external conditional stimulations provoking a process of inner inhibition, i.e. that sleep in an active process of inhibition in the cerebral cortex (Pavlov, 1926). Data of electro-encephalographic investigations of natural sleep in animals speak in favor of this (Tskipuridze, 1950b; Kogan, 1953, 1955).

As for the reaction of awakening, then, first, the conclusion that the generalized reaction of the cortex under the effect of external stimulations sets in exclusively because of the diffuse effect from the subcortex, and does not arise as a result of the arrival of afferent impulses to a given projection territory of the cortex, is in contradiction with the following observation. In non-narcotized dog under the influence of photic stimulation the reaction of depression of slow potentials all over the cortex arose; after isolation from one another by section of optic, auditory, and motor zones of both hemispheres, photic stimulation provoked the reaction of depression only in the optic zone (Tolmasskaia, 1949). Secondly, the conclusion that the awakening of the animal under the effect of external stimulations occurs exclusively because of diffuse "impulsation" from the reticular formation and that the arrival of afferent impulses

through specific afferents to certain projection territories of the
cortex does not thereby play any special role, is found, as Bremer
notes, in contradiction with the following fact, which has already
been cited once. After bilateral stimulation of the auditory projection
cortical territories, sound stimulations stop arousing the animal
(Bremer and Terzuolo, 1952, 1954).

This cardinal fact, that the arousing of a normal animal occurs
under the influence of such stimulations as cause an oriented reaction
(Beritov, 1932), completely ignores the authors' concept that arousing
under the effect of external stimulations occurs exclusively because
of diffuse activation of the cortex from the reticular formation.
Accordingly, in normal animal generalized reaction of depression of
the slow potentials in the cortex usually also sets in under the
influence of such stimulations as cause oriented reaction. However, it
is impossible to consider the arousing as the direct consequence of
the arrival of afferent impulses to the cerebral cortex. As known,
in normal animals oriented reaction occurs through the cortex. In
each of the cortical analysors there are efferent projection neurons,
the axons of which proceed downward into the spinal cord and medulla
oblongata. At the effect of external stimulation of a certain intensity
in the corresponding analysor there occurs, together with excitation
of the different neurons with short axon and excitation of the association

pyramids, the excitation also of projection neurons stipulating

realization of the oriented reaction, i.e. movements of the head,

ears, and eyes, intensification of the respiration, etc. (Beritov,

1948; Bremer, 1952). It has been demonstrated that the arousing

begins under the effect of the setting in of an oriented reflex,

with the realization of which a whole series of stimulations is

connected, of which stimulations of the labyrinths and of the

proprioceptive muscles of the neck have chief importance (Beritov,

1932). Thus, the reaction of the arousing is connected with

primary activation of the cortex.

In this respect the facts obtained by Bremer and Terzuolo

(1954) are conclusive: electrical stimulation (of moderate intensity)

of different parts of the cerebral cortex provoked the awakening of

the animal with its characteristic external phenomena and with the

characteristic changes of the electrical activity of the cortex

and subcortex; this reaction arose especially easily at stimulation

of the sensory-motor territory of the cortex.

All these data show that the subcortex, particularly the

reticular formation of the stem of the brain, does not regulate

the activity of the cortex. Its role consists of maintaining the

tone of the cortex and of the definite degree of its excitability,

as Pavlov computed.

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At the present time not only the phenomena of sleep and wakefulness, but also the phenomena of consciousness and perception, are linked by American physiologists with the activity of the subcortical formations, primarily of the midbrain (Bernhaut and coworkers, 1953; Gellhorn and coworkers, 1954). In this respect particularly characteristic is Penfield's concept. In 1938 Penfield set forth a hypothesis, according to which the highest level of integration is found in the midbrain and not in the cerebral cortex. In 1950 and in 1952 this position was already promoted as a theory of localization of consciousness in the midbrain (Penfield and Ramussen, 1950, 1952).

In Pavlov's laboratories it was demonstrated that in higher animals, after removal of the cerebral cortex, all conditioned-reflex activity was lost and new conditioned reflexes did not form. Thus, the midbrain discloses no visible capacity for the formation of conditioned connections. Hence, an animal after removal of the cortex, possessing according to Penfield "higher levels of integration", proves entirely devoid of "psychism", only inborn reflexes are preserved in it, and after hundreds of associations elaboration is possible for just the simplest reactions of adaptation (Popov, 1953). As said, at removal of the cortical end, for instance, of the auditory analysor, conditioned-reflex activity to auditory stimulations becomes impossible.

The facts obtained by Penfield at stimulation of the cerebral

cortex in man can serve as a good argument for this, that consciousness is connected, namely, with the activity of the cerebral cortex, but then just such a conclusion should be made on the basis of the fact that direct electrical stimulation of the surface of the cortex can regularly provoke one or another perception.

(End of Part I)

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