THE INTERACTION OF NEURONES[1]

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That neurones interact is obvious from the simple consideration that each of you has several billion neurones in your cerebral cortex, yet remains a single personality. The general question I wish to raise is: How is this unification brought about? The existence of gross interaction is shown, more precisely, in many ways.

Lashley (1929) has offered impressive evidence of a mass function of the brain—not alternate to, but superimposed upon, its specialized local function—by studying the learning capacity of rats after the production of various brain lesions. The deterioration of learning ability he found to be highly correlated with the amount of cortex lost (r up to 0.86); but, since extensive criss-cross slicing of the whole cerebrum or removal of sufficient occipital lobe to destroy the same actual volume of cortex as destroyed by the cuts led to like learning losses; this did not correlate with the extent of interruption of recognized anatomical connections. What sort of neurone interaction might lead to this mass response?

Again, in the course of establishing a conditioned reflex, a particular afferent system comes to exercise control over an efferent oneup on which it normally has no action. In neurological terms, this means that two brain centers become able to interact physiologically as a consequence of having been repeatedly set into action together. How does the repeated ringing of a bell, when food is offered, open pathways between the auditory system and the salivatory nucleus?

Another form of interaction is manifested in the synchronized electrical beating of large numbers of neurones. This is widely manifest in neural masses—from the synchronized discharges of the uniformly illuminated retina (Adrian and Matthews, 1928), or the like impulse trains set up from the two respiratory centers and recorded in the phrenic nerves (Gasser and Newcomer, 1921), to the regular alpha rhythm of the human occipital cortex, and the equivalent regular beat of the isolated frog olfactory bulb (Libet and Gerard, 1939). How is this interaction achieved?

Closely related is the problem of the slowly spreading cortical waves seen in epilepsy or induced by convulsant drugs (Gibbs, Lennox and Gibbs, 1936; Adrian, 1936; Gerard and Libet, 1940). Even the well recognized dominance by more rostral neural masses, which check or suppress the activity of more caudal and phylogenetically older ones, presents a similar problem in interaction on a gross scale.

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At a finer level, a great mass of neurophysiological research has demonstrated in some detail the ability of one neurone to act upon another to change the state of the second (e. g. Symposium, 1939). This may be made to discharge and after-discharge or to cease action already in progress; its threshold may be lowered so that subsequent stimuli are facilitated, or raised so that they are suppressed; its potential may be shifted in the negative or in the positive direction; its metabolism may be increased or diminished. How are these effects brought about?

The mechanism of interaction, in this last case, is clearly that of synaptic transmission; and this of course analyzes further into chemical or electrical mechanisms. Some years ago (1932) I wrote, "Conduction along a nerve fiber involves excitation of a resting region by an active one, and both electrical and chemical components are present in the mechanism of propagation. At the ending, which is specialized, at least anatomically, either or both components might well be exaggerated to facilitate conduction over a critical region. Long enduring action or depolarization potentials or special chemical accumulation might equally well be utilized in various situations." To my mind, the enormous increase of information during the interim has not invalidated this position.

I do not propose to discuss the exciting controversy between those who insist on a material or humoral mechanism and those who equally insist on an energetic or electrical one. The chemical theories have faced a major difficulty, in accounting for very rapid changes, by emphasizing the appropriately high concentration of enzyme in junctional regions. The electrical ones have been similarly bothered by the problem of slow and enduring changes, and have interpreted these in terms of maintained interneurone bombardment and of long after-potentials. Since there is no anatomical basis for the former in autonomic ganglia, yet brief preganglionic tetany can alter the activity of ganglion cells for minutes afterwards (Bronk, Symposium), interneurone activity cannot be a universal explanation. On the other hand, it has long been known (Gerard, 1930), though often overlooked, that a few seconds tetanus may leave, even in nerve, considerable after-potentials which actually increase in magnitude during three or four minutes and endure for over fifteen.

Aside, however, from any chemical or electrical changes set up in highly localized fashion at a synapse, there is good evidence that each of these mechanisms acts more generally upon neurones, and independently of synaptic transmission. That the physico-chemical milieu of nerve cells can profoundly influence their activity or responsiveness, is now widely recognized. One need only recall, for example, the ability of an increased blood potassium concentration to arouse cats from anesthesia (Dubner and Gerard, 1939), or to triple the duration of the rebound limb positions following electrical stimulation of the deep cerebellar nuclei (Gerard and Magoun, 1936). I shall, therefore, in the remainder of this address, emphasize rather the electrical mechanisms which act grossly on the individual neurone and which are of prime importance in the interaction of the many.

Only in recent years has such non-synaptic electrical interaction begun to receive the attention it has long and obviously merited (e. g. Gerard, 1931). Now much evidence of its importance is appearing in the literature. Some examples are: the ability of a nerve action current to modify the excitability of a resting stretch of nerve even when the impulse with which the potential is associated is prevented by block from reaching this resting stretch (Hodgkin, 1937; Lorente de No, Symposium); the even more striking ability of the action currents from above such a block to re-initiate an impulse in the same nerve fiber two millimeters distant (Blair and Erlanger, 1939; Tasaki, 1939); the similar ability of the currents from one active nerve fiber to alter the thresholds of adjacent ones (Katz and Schmitt, 1940); and the tendency of separate axons in an isolated nerve (Adrian, 1930), of separated nitella cells lying in the same dish of water (Hill, 1939), and of individual spermatozoa clustered about a single egg, (Lillie, 1932), gradually to synchronize their rhythmic actions. I shall, however, base the further discussion on experiments carried out during recent years by Dr. Libet and myself on the isolated frog brain.

The frog brain is a peculiarly favorable preparation for study of the electrical activity of neurones. Its cells maintain the *in vivo* rhythm, some six beats a second of thirty microvolts amplitude, for several hours after the complete isolation of the cerebrum. Indeed, the potentials are commonly larger and more regular *in vitro* than they were in the same brain in the unanesthetized frog, exposed earlier under ether. Even a tiny bit of brain, as little as 1/10 of a milligram carefully teased from the olfactory bulb, can maintain its regular rhythm. Obviously, no circulation changes can confuse the picture, and the supply of oxygen and other substances by diffusion is adequate. It is thus possible to vary, in controlled quantitative fashion such conditions as temperature, salt or drug concentrations, or the like, while keeping other variables unchanged. It could be shown, for example, that a rise in temperature increases not only the amplitude and the frequency ($Q_{10}=2.3$) but also the regularity of the rhythm; while potassium increase, though accelerating the rhythm, soon disrupts it; and calcium, as usual, has the reverse action.

Further, the cells in the frog hemisphere are arranged in relatively simple patterns and form what amounts to a mosaic sheet near the ventricular surface. They are easily accessible to electrodes at either cell pole and thus, as we shall see, simulate the membrane of a nerve fiber studied by internal and external leads. Synaptic transmission in the isolated brain is retained under ordinary circumstances. Thus, brief tetanization of the olfactory nerve leads to an increase in amplitude of the olfactory bulb rhythm and to the appearance of waves at a higher frequency, and these changes may endure for several minutes.

Along similar lines, Mr. Tokaji and I have recently found that brief soaking of the brain in either acetylcholine or in eserine, at a concentration of 1 part in a hundred million, leads to a gradual increase (the maximum effect is not attained until a half-hour after the soaking) in the continuous activity and to the appearance of characteristic large diphasic repeated waves, Fig. 1. It is not yet certain, of course, that these drugs

act here to enhance synaptic stimulation, but their low effective concentrations, and the ability of nicotine to abolish their action, strongly suggests this.

Finally, the neurone mass in this preparation shows striking synchronization. That the few hundred or thousand cells in a single olfactory bulb are beating together is made strongly probable from the mere existence of the ordinary rhythm; for, if each cell were active independently of the others, a large, smooth and regular potential wave could not result. The synchrony is made quite certain when this ordinary rhythm can be altered, by appropriate change in conditions, so that regularly repeated waves occur at frequencies between one and fifty a second, at amplitudes from a few microvolts to over two millivolts, and with shapes from almost pure sine waves to highly skewed, humped,

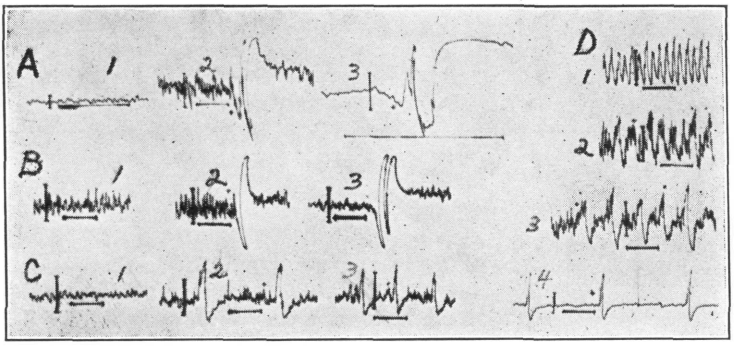

Fig. 1. Action of acetylcholine and eserine on isolated frog brain. Records in three horizontal groups (A, B, C) from three hemispheres, in vertical group (D) from olfactory bulb. Horizontal line = 1 second (read left to right), vertical line = 100 μV, except as indicated.

A 1. Normal (30μV); 2. 25 min. after 3 min. in 10^{-6}% eserine (HCl); 3. soon after 2, faster (300μV).

B 1. Normal; 2. 11 min. after 3 min. in 10^{-6}% acetylcholine; 3. 4 min. later (300μV).

C 1. Normal; 2. 24 min. after 3 min. in 10^{-6}% acetylcholine; 3. 9 min. later.

D 1. Normal; 2. 8 min. after 3 min. in 10^{-6}% acetylcholine (in serum); 3. 17 min. later; 4. 13 min. after 3 (300μV).

and even polyphasic, spike-like profiles. Though such irregular waves could be the resultant of simple waves of individual cells, so timed in their appearance as to sum to the recorded wave shape, this is *a priori* unlikely and only possible at all on the basis of recurrent excitation circuits. Yet nicotine, which blocks such circuits, still leaves very regular skewed waves.

Cell synchronization is, therefore, present and can profitably be studied in the isolated brain. Thus, greater regularity should denote more perfect synchronization, and it should be, and is, more difficult to disrupt a more regular rhythm than a less regular one by desynchronizing agents, such as potassium. By such criteria it has been shown

that synchrony is enhanced by an increase in temperature or calcium; by certain drugs, notably caffeine or nicotine; by a diminution of potassium or sodium; and, especially, by maintained constant currents.

The most useful property of this isolated brain, however, is its ability to generate slowly traveling waves; evoked especially conveniently by a brief soaking in caffeine. Following this treatment, there appear large surface-positive waves, of 0.1 to 0.2 second duration and one or two millivolts potential, each of which passes over into a surface-negative potential of lower amplitude and several times longer duration.

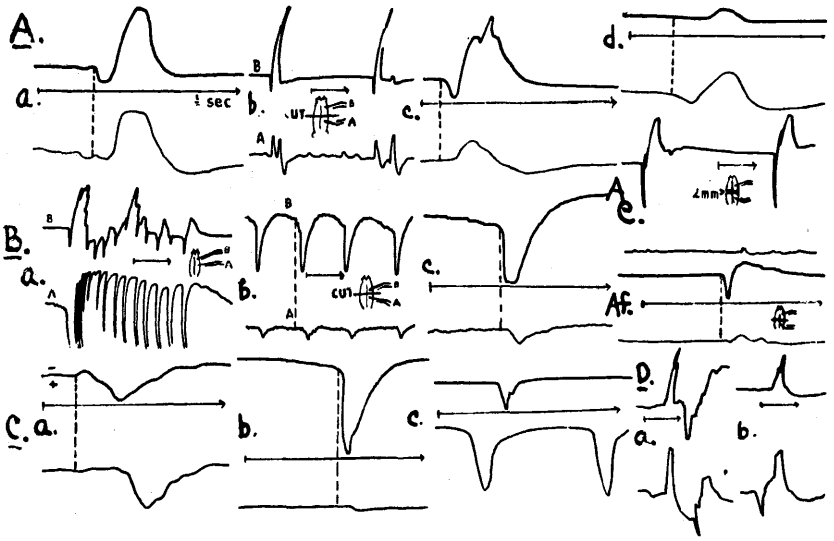

Fig. 2. Interaction across cut. Two channel simultaneous records.

A. a. Caffeine waves before cut; b. after complete transection across hemispheres, recording from each piece as shown; c. same later, showing front piece originating wave; d. still later, showing hind piece originating wave; e. pieces separated by 2 mm. (connected by Ringer's), note lack of interaction; f. pieces reapposed.

B. Another brain; a. caffeine waves before cut; b. after complete transection; c. same, faster speed, note that wave clearly spreads with finite velocity across cut.

C. Another brain; a. caffeine waves before cut; b. after complete transection between the two sets of electrodes; c. later, independent action of the two halves, showing non-interaction of recording systems.

D. Another brain; a. caffeine waves before cut; b. after cut.

The main wave, arising from a flat base line, is commonly followed by a train of satellite waves of progressively greater amplitude and lower frequency until the whole train stops. The entire sequence is repeated at regular intervals. Two channel recording shows that these caffeine waves travel over the hemisphere, most commonly in a rostro-caudad direction but sometimes in the reverse one, at the usual speed of 5 centimeters a second. (Fig. 2).

This slow propagation does not depend on synaptic excitation of successive cells, with or without interneurone circuits. The spreading caffeine waves are not altered by application of nicotine, which blocks synaptic responses, such as the on and off light response of the optic lobe, even in the presence of strong caffeine. Even more conclusive (since it might be argued that nicotine failed to block interneurone synapses which we could not directly test) is the finding that the caffeine waves can continue their longitudinal travel across a complete sharp cut made transversely through the brain at the middle of the hemisphere, providing the two brain halves are accurately reapposed. Separation of a fraction of a millimeter, even with Ringer solution in the

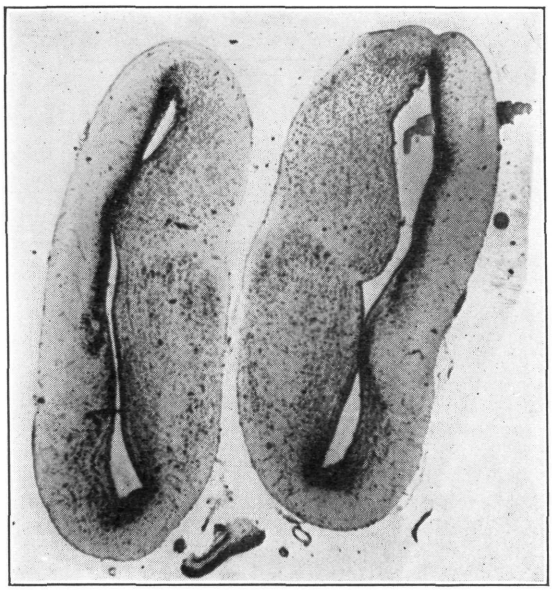

Fig. 3. Coronal section through frog hemispheres. Microphotograph ×15. Position of ventricular and pial electrodes indicated on right upper pallium.

gap, stops the wave at the cut; reapposition again allows it to travel from olfactory bulb to occipital pole, with no significant disturbance at the interposed cut. (Fig. 2).

Clearly, in this case, synaptic conduction is impossible; and a chemical mechanism cannot come in question. The probability was therefore strong that intercellular electric currents supply the mechanism for this wave propagation. If correct, it would follow that the activity of neurones in the normal brain, as well as in the caffeinized one, should be modified by passing appropriate constant currents through the brain mass—that is, by polarizing it—and, indeed, ascending or descending currents, of .05 milliamperes or less, do profoundly affect the electrical activity of the isolated hemisphere.

It will be useful at this time to present a theoretical interpretation in the light of which further data can be evaluated. The cerebral neurones, as can be seen in Fig. 3, form a sheet of closely packed cells near the ventricle and in a plane parallel to the pial and ventricular surfaces. Each cell is oriented with its dendrites towards the pia and its axon towards the ventricle. Let us assume that, in addition to the well recognized membrane potential, each cell is normally polarized along its perikaryon or soma, so that the dendritic pole is negative to the axonic. As seen in Fig. 4, the cell sheet would thus resemble a polarized membrane, as the nerve membrane, with an electric double layer. The postulated direction of the potential, as regards the outside and inside of the hemisphere is, of course, the reverse of that in the nerve membrane and, if correct, it should follow that an- and catelectrotonic effects produced by constant currents should also be inverted relative to the brain surface. We shall see later that this is the case.

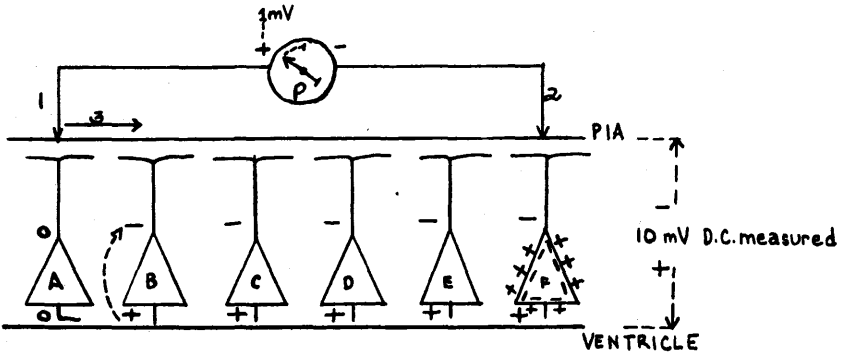

Fig. 4. Schema of cell layer in frog's cerebral hemisphere. See text. A caffeine wave is spreading as indicated by arrow 3 and has just discharged the somatic polarization of cell A. Cell B is discharging. The amplifier shows a pial surface positive wave of 1 mV. The usual interior-exterior polarization of the neurone surface is shown for cell F.

We postulate further that when the somatic potential is sufficiently decreased, that is when the axonic end of the cell moves in the direction of negativity relative to the dendritic, the cell becomes active and the somatic potential is discharged. Such a discharged cell, failing to oppose by its own potential the currents which tend to flow through and about it from the charges on neighboring cells, would act comparably to a depolarized region of the nerve membrane. The lowered resistance at this spot would permit adjacent somatic potentials to discharge, the neighboring cells would become active and depolarize, and a spreading wave of cell depolarization and activity would result. Similarly, one cell might actively send current through another with equivalent results. Since activation of one cell by another does not depend on synaptic transmission but on currents flowing through intercellular fluid, nicotine should not stop them, nor should a sharp cut providing the cut surfaces remain well apposed.

Many consequences of such a theory can be directly tested in terms of concrete predictions. For example, there should be a steady potential difference recorded between electrodes placed on the pial and ventricular surfaces of the thin hemisphere, the pial surface being negative to the ventricular. Measurements show a consistent DC potential, normally of 2–3 millivolts magnitude but with occasional much higher values, and usually in the right direction. The direction of this potential sometimes shifts spontaneously and can easily be altered by deliberate polarization across the thickness of the hemisphere. When traveling caffeine waves are then elicited, it is found, as the theory predicts, that the main wave has also reversed its polarity. In fact, as Fig. 5 shows,

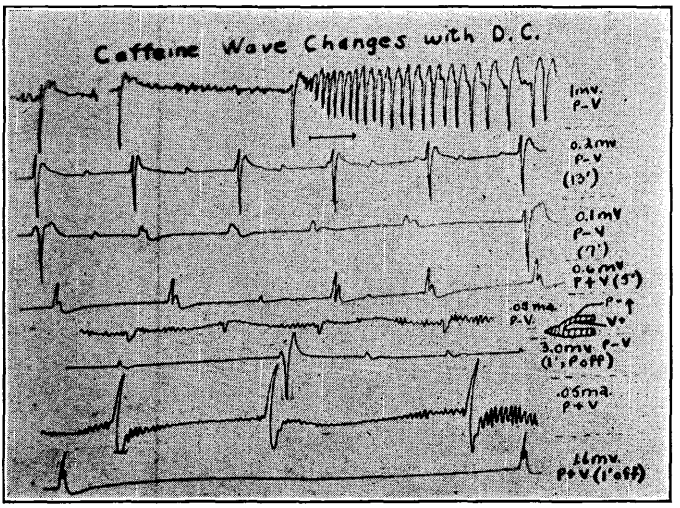

Fig. 5. Change in caffeine waves with P-V potential. From above down, time intervals between records are: 13 min., 7 min., 5 min. At 5th line, polarization is in progress as indicated, then 1 min. later. At 7th line reverse polarization in progress, then 1 min. later.

the direction and configuration of the caffeine wave correlates very well with the simultaneously measured P-V potential (pial-ventricular) as this slowly shifts in magnitude and direction.

The theory also predicts that the caffeine waves, as measured between electrodes on the pial and ventricular surfaces of the hemisphere, should be larger than those recorded between two electrodes on the pial surface, and should always be simple in form even when the latter show polyphasic variations. These predictions have also been experimentally confirmed, the P-V recorded wave being regularly larger, sometimes showing two or more times the voltage of the surface recorded wave.

Polarization through the hemisphere's thickness, ventricle negative to pia, would tend to depolarize the somatic potential, and therefore

should act comparably to cathodal polarization of a nerve. This it does, for it will regularly initiate waves in an otherwise inactive brain, and seems to cause the locus of origin of caffeine waves already present to shift towards or to the region of depolarization. "Anelectrotonus" has the inverse actions. Even the intensity and duration of the cell after-potentials are altered by polarization in a manner comparable to those of nerve.

It is thus obvious that this theory of somatic potential and intercellular electric currents, so closely analogous to the membrane theory of nerve conduction, serves well to explain the slowly spreading caffeine waves—as indeed it should, since it was devised for this purpose.

What is more important is its ability to explain other phenomena, as those of neurone interaction cited at the start of this address, without the necessity of introducing additional assumptions. Thus, for example, the spontaneous electrical beat recorded from brain neurones is probably a rhythmic variation of the somatic potential—indeed, if the membrane potential were to change simultaneously over the entire cell surface no external potential variation would appear—and the synchronization of the many beating cells follows simply from the above picture. Just as the current from a pacemaking nerve fiber or a nitella cell is able to sweep into unison with it other nearby units, about to fire off spontaneously in the course of their own rhythmic activity, so a pacemaking neurone (and not necessarily the same one at all times) would engage adjacent ones. Instead of having a slowly spreading wave, because the currents from one cell require a short time for their "detonator" action to stimulate the next, there would result an essentially simultaneous activity of adjacent units as their individual spontaneous rhythms fall into synchrony. The unifying action would spread rapidly to more peripheral units as the number of cells beating together increased and ever larger currents consequently flowed through the brain mass.

In the same way, by the interaction and summation of intercellular currents from large numbers of neurones, we can account for the phenomenon of a mass action independent of synaptic connections and for the existence of dynamic patterns of brain activity superimposed on, and partly independent of, the anatomical architecture. It is also obvious, I think, without developing the argument in detail, that the simultaneous activity of two nearby cortical regions, as in the course of conditioning, would alter the potential fields which would accompany the activity of either region alone and could so lead to the establishment of new functional patterns, of new conditioned reflexes. Finally, the somatic potential theory lends itself to a simple explanation of central inhibition which I shall shortly develop.

It will be desirable now to turn your attention again to the various interpretations of the functioning of the central nervous system which have been built up from the known properties of nerve fiber and synapse action, especially by Gasser and by Lorente de No. Gasser's theory of inhibition (Gasser, 1937), depending on the capture of an interneurone by one active neurone chain and so blocking transmission in a second chain to which it is also necessary, is familiar to you. I have dia-

grammed a slightly modified version of this in Fig. 6, which includes also a simple extension of the reasoning to account for the phenomenon of conditioning.

Of course, such nerve impulse theories fit admirably the phenomena of summation, facilitation, after-discharge, and the like—in connection with which they have been developed—and I daresay they could be extended to account for the phenomenon of synchronization, though this would certainly involve difficulties. But it is important to note that all such interpretations rest heavily on very precise structural relations between neurones, especially on the number and distribution of synaptic end-feet, and on the no less precise timing of the arrival of excitation from different sources so that they can or cannot sum their

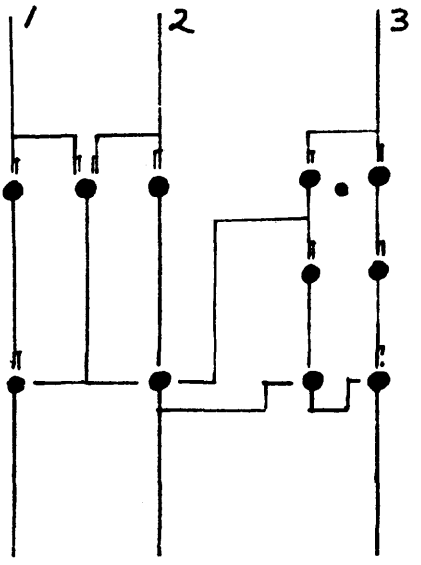

Fig. 6. Assume two synaptic endings on a neurone must act together to produce excitation. Then an impulse in afferent fiber 1 or 2 will excite its appropriate efferent. But 1 inhibits 2 by capturing the common interneurone. When 1 and 3 are active together, 1 can discharge the efferent neurons of 3.

effects, as the case may be. And, despite the clear experimental demonstration of many of these necessary conditions, there remain certain serious difficulties to face.

The facts outlined earlier, for example, that block of synaptic excitation by nicotine or of nerve fiber conduction by anatomical section do not abolish neurone synchrony or the spread of slow waves, are not reconcilable with pure nerve-impulse theories. Similarly for the equal loss of learning capacity, with equal loss of cortical tissue, with rat cortex destruction, independently of whether or not nerve pathways have been extensively interrupted. A further problem is encountered in interpreting the effects of influent nerve impulses

reaching different parts of a single neurose. It does not seem probable that groups of end-feet synapsing on widely separated dendritic branches can produce effects which interact, either for summation or inhibition, by any sort of membrane-propagated change along the neurone in question. Not only would a propagation of graded intensity, rather than of all-or-none character, be necessary, but also qualitatively different ones for excitation and for inhibition would be needed.

This latter difficulty would not arise if all the phenomena of inhibition could be accounted for by the shift of interneurones from one pathway to another. But such an interpretation does not seem able to account for all these phenomena. To take just one: ganglion cells, which are kept in a state of inhibition by maintained stimulation of inhibitory nerves to them, not merely fail to increase their resting rate of metabolism, but actually show a very considerable diminution in respiration (Dann and Gardiner, 1930). No type of theory which accounts for inhibition by any kind of increased activity, can be reconciled with these facts. Rather, each neurone itself, under the influence of whatever factors, must change its state in a direction inverse to that of excitation.

Finally, I must mention a body of data which throws some doubt on the interpretations of neurone function which require precise and set anatomical connections. Weiss (1940) has recently made separate implants under the dorsal fin of amblystoma of an extra leg and of a piece of spinal cord from another salamander. Ninety per cent of the neurones in the cord fragment disappear, and the remainder become completely disorganized by migration and growth of new processes. Dorsal column cells, for example, may sprout fibers which run out from the graft to form motor and sensory connections with tissues of the host and, particularly, with the neighboring limb implant. Yet, as the limb becomes innervated, it shows at first repeated generalized contractions, indicating synchronized discharges from many neurones, and later there appear well defined reflex responses which possess the usual attributes; of spacial and temporal summation, of gradation of response with stimulus intensity, of after-discharge, and the like. Inhibition has not been observed. The extent of synchronous action of these morphologically disturbed neurones is particularly well illustrated by implanting two legs on opposite sides of the cord section; for in such a preparation the two limbs always respond together.

In terms of the somatic potential, a theory of inhibition requires one essential assumption. If excitation follows the decrease or disappearance of this potential, inhibition should result when it is enhanced. Just as diminished polarization is associated with increased metabolism, increased polarization, produced from without, should lead to a decrease. We have in fact some evidence on nerve, although it is far from decisive, that a cathodally polarized region increases, an anodally polarized one decreases, its respiration. The further assumption needed is that those synaptic endings which excite a cell tend to be grouped at one pole, while those which inhibit it are grouped at the other. Then an identical action, such as the production of a local negativity, would, in the case of

an excitatory synapse oppose and so diminish the somatic potential, in the case of an inhibitory synapse sum with and so increase it.

To what extent such an anatomical disposition of functionally different endings does exist in the central nervous system is unknown, but that it can exist is clearly shown by the case of the Mauthner cell in certain fish brains. This large single cell, with a soma well over a millimeter long and two dendrites extending through the thickness of the tegmentum, receives synaptic connections of several well recognized types and from a number of known pathways (Bodian, 1937). (Fig. 7). End-feet on the axone hillock are of the small button type, come from fibers in the posterior longitudinal bundle, and are supposed to produce inhibition. End-feet on the dendrites are of the club type, come from the acoustic system, and are believed to cause excitation.

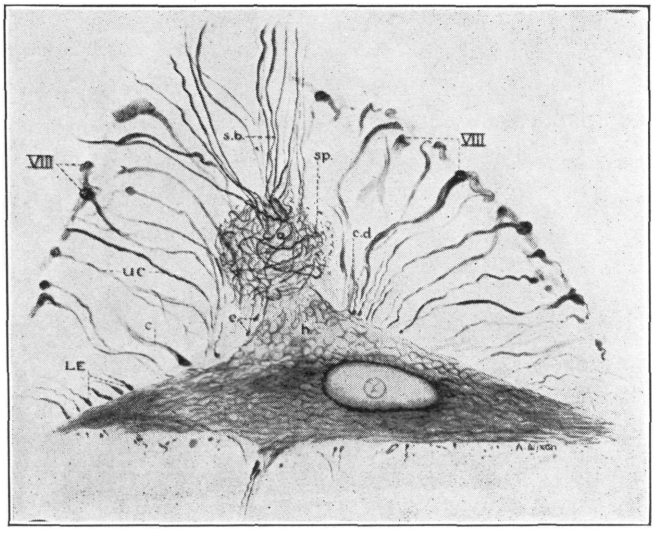

Fig. 7. Mauthner cell of the gold fish brain (from Bodian). Synaptic endings on the axone and axone hillock (above) are anatomically distinct from those ending on the soma or dendrites. This affords an anatomical basis for the type of inhibition here suggested.

The evidence, then, seems to show that nerve impulses traveling along definitive pathways are important in controlling the interaction of neurones, especially as they combine with one another in producing the special patterns of motor discharge and other fine regulations; and that many chemical substances, brought in the blood stream or formed locally, are likewise able to act upon neurones, regionally or generally, to modify their actions. But, in addition, there is the third factor, of electric currents flowing through and between neurones, which, perhaps more than either of the others, is involved in the interactions of nerve cells in larger groups and masses and which may also play an important role in certain of the more specific actions of one cell on another.

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