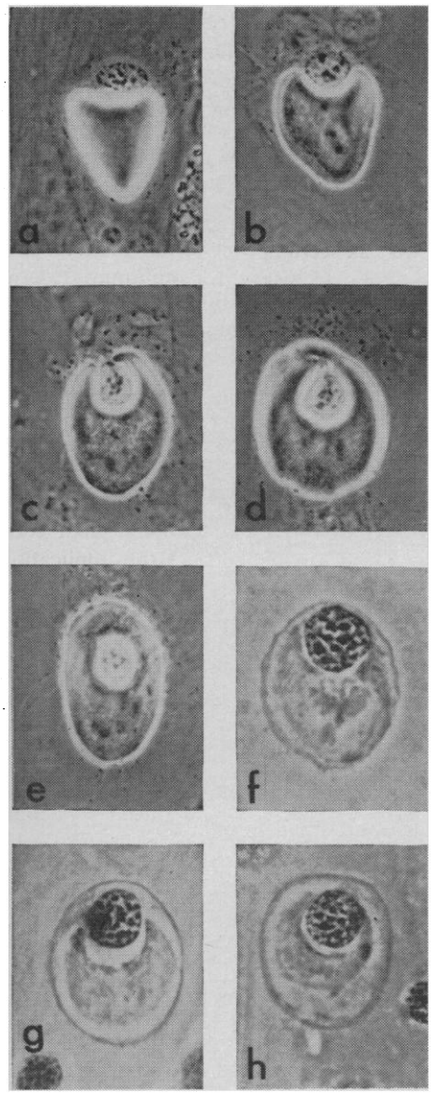

Fig. 2. Cell-crescent body associations representing a graded series in *Gromphadorhina* hemolymph (× 760). *a–e*, Phase-contrast studies of stages representing phagocytosis. *f–h*, Cells, stained with gallocyanin-chromalum, showing progressive decrease of inner crescent substance.

extensions are frequently observed in the final phase of cell ingestion. An example of this is shown in Fig. 1*c*. Were this an extrusion phenomenon, one might expect to see cytoplasmic swelling and evidence of a ruptured membrane.

In the majority of cases, associated bodies in the hemolymph are juxtaposed in such manner that the anucleate member of the pair (averaging 25 μ in diameter and 5 μ at the thicker edge) partly surrounds the smaller nucleated member (averaging 8 μ in diameter) with broad pseudopod-like extensions. The anucleate cytoplasm thus has the shape of a crescent (Fig. 2*b*). In these associations, approximately half of the partially ingested cell, which is round, flat, and scanty in cytoplasmic content, projects beyond the two "pseudopodia" forming the lesser curvature of the crescent body. In other associated pairs either "pseudopod" extensions have not yet formed, or the cell-juxtaposed face of the crescent body is no more than a slight concavity (Fig. 2*a*). The variety of associated pairs seen suggests an ordered sequence resembling phagocytosis (Fig. 2, *c–e*).

Once incorporated within the cytoplasm of the crescent body, the nucleus of the former cell persists in a somewhat enlarged state. Newly formed cells of this type may be seen to undergo breakdown of the inner crescent form (Fig. 2, *f–h*)—an area of more dense granulation within the crescent body which conforms to the shape of the cytoplasmic membrane without contacting it. Fixed and stained cells are represented in Fig. 2 (*f–h*) because of enhanced contrast afforded in stages which are apparently participating in the reorganization of the inner crescent substance.

When tested by the periodic acid–Schiff (PAS) reaction, the crescent body substance was strongly positive and the intensity of staining was almost uniform. Negative results were obtained with the diastase reaction. Lipid material could not be demonstrated with Sudan Black B. Staining procedures selective for nucleic acid identification (Feulgen, gallocyanin-chromalum, toluidine blue, pyronin Y, acridine orange) gave negative results. Stains were used with various fixatives and with ribonuclease.

What is the fate of newly formed cells? If the degree of PAS sensitivity is indicative of high polysaccharide content, then one might regard this feature as representing an energy store available for the possible fulfillment of subsequent cytological events. But evidence for functional integration between the ingested nucleus and its adopted cytoplasm is lacking, even though cellular disintegration does not seem to occur. Another unsolved question relates to the origin of the phagocytic crescent bodies.

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Cross-Correlation Analysis of Electroencephalographic Potentials and Slow Membrane Transients

Abstract. *Cross-correlation analysis reveals a close correlation between the waves in an electroencephalogram and slow membrane transients of single neurons of the sensorimotor cortex of cats during spontaneous activity, augmenting and recruiting responses, and after local application of strychnine. Time-series correlation coefficients up to 0.7 have been computed. It is suggested that the waves of the electroencephalogram reflect an integration of the changes of membrane potentials in both the cell bodies and dendrites of cortical neurons.*

In addition to spike activity, intracellular recordings of the electrical activity of neurons reveal slow changes of the membrane potential which are referred to as postsynaptic potentials. Similarities in the time course of afterpotentials of motoneurons and the periodicity of α-waves in the electroencephalogram (EEG) had led to the assumption (i) that the EEG consists of a summation of postsynaptic potentials (*1*). Because of the characteristics of nonrefractory and graded responses it was assumed (ii) that the EEG originates in the apical dendrites or might be a summation of dendritic postsynaptic potentials (*2*). Other theories of the origin of the EEG consider (iii) soma-dendritic dipoles, (iv) autorhythmicity of neuronal elements, (v) modulation of cortical d-c potentials, and (vi) activity of glial cells to be the important factors (*3*, *4*).

The striking similarities in the time course of EEG potentials and slow membrane transients, particularly de-

Table 1. Average of decay-time constants. Results expressed in milliseconds (PSP, postsynaptic potential).

Neuroelectric activity	n	Membrane transients	EEG potentials
Depolarizing PSP's and EEG waves			
Augmenting	20	7	10
Recruiting	20	14	13
Strychnine-induced waves	15	13	17
Hyperpolarizing PSP's and EEG afterpotentials			
Augmenting	10	53	38
Strychnine-induced waves	15	93	116

polarizing and hyperpolarizing potentials or afterpotentials, have been qualitatively shown by several investigators (i) in hippocampal neurons during seizures (*5*), (ii) in thalamic and cortical neurons during recruiting (*6*), (iii) during caudate spindle waves (*7*), (iv) in neurons of the visual cortex during spontaneous activity (*8*), and (v) in a stratigraphic EEG analysis (*4*).

We have analyzed quantitatively the correlation between EEG waves and slow membrane transients of single cortical neurons. By computing the cross-correlograms $\Phi_{eeg,membrane}(\tau)$ and the time-series correlation coefficients $\rho_{eeg,membrane}(\tau)$ this correlation was statistically confirmed. Synchronous recordings of the surface EEG and of the intracellular potentials of 50 selected neurons were obtained by standard microelectrode techniques from 23 cats lightly anesthetized with pentobarbital sodium. Most of the neurons were situated about 600 to 1500 μ beneath the surface of the sensorimotor cortex corresponding to layers III and V.

Test stimuli to both the nuclei ventrooralis posterior thalami and the centrum medianum, and also to the bulbar pyramidal tract for identifying the cortical neurons with antidromic responses as pyramidal tract cells, were applied by means of bipolar electrodes.

The recording system consisted of micropipets filled with 2*M* potassium citrate, a cathode follower, a direct-current amplifier for the membrane potential, RC-coupled amplifiers with the same time constants of 100 or 250 msec for membrane transients and EEG waves, oscilloscopes, a continuous strip-recording camera, and a multichannel frequency-modulated magnetic tape recorder.

Membrane transients with spikes omitted and EEG waves of 40 recordings were digitized with Mnemotron equipment at sampling intervals of 2, 5, 10, or 20 msec for periods as long as 4 seconds, and were subsequently processed on an IBM 7090 data processing system (*9*). Samples of the types of recordings used for the computation of the correlograms of the evoked responses are shown in Fig. 1. The patterns for strychnine-treated preparations (Fig. 1, *A* and *B*) are typical for those occurring at the beginning of strychnine seizures after topical application of 1-percent strychnine sulfate. The pattern for the augmenting response is typical for EEG responses in the middle of a train of stimulation of the specific thalamic nucleus at about 5 cycles per second (*10, 11*).

These EEG patterns were usually accompanied by synchronous discharges of the cells, as shown in the lower records of Fig. 1, *A–C*: a depolarizing potential with burst discharges, followed by a long-lasting hyperpolarizing potential of 150- to 200-msec duration. Moreover, Fig. 1, *A* and *B*, show that after strychnine poisoning neither the duration nor the amplitude of hyperpolarizing potentials are diminished, whether after spontaneous strychnine-induced waves (Fig. 1*A*) or after waves elicited by thalamic stimulation (Fig. 1*B*), when compared with corresponding potentials in unpoisoned preparations (Fig. 1*C*) (*12*). In fact, hyperpolarizing potentials obtained under those conditions frequently have a longer duration and greater (decay) time constants (Table 1) than normal ones.

The correlograms (*13*) of these evoked patterns, and in addition of evoked recruiting patterns, are shown in Fig. 2*A*. The ordinate scale in Fig. 2*A* is normalized to −1.0 for the highest value of the correlogram for the strychnine-treated preparation. Note the variability of the augmenting responses shown in the two different traces, and the similar time course of strychnine-induced and recruiting patterns. The similarities between the patterns for strychnine-treated preparations and for recruiting, and the nearly identical (decay) time constants (Table 1), support the notion that the increased excitability after strychnine poisoning may be localized at dendrites (*14*), since the

Table 2. Characteristics of 40 correlograms. The distance between the microelectrode for membrane transients and the macroelectrode for the EEG was 2 to 3 mm.

Neuroelectric activity	n	Phase lead of negative max. (msec)	Cross-correlation coefficient of positive and negative maximum
		Spontaneous	
Normal	17	0 to 60	+0.1 to + 0.5 − .2 to − 0.5
Strychnine-induced	10	10 to 60	+ .1 to + .7 − .3 to − .7
		Evoked	
Augmenting	8	8 to 32	Not computed
Recruiting	3	0 to 48	Not computed
Strychnine-induced	2	8 and 12	Not computed

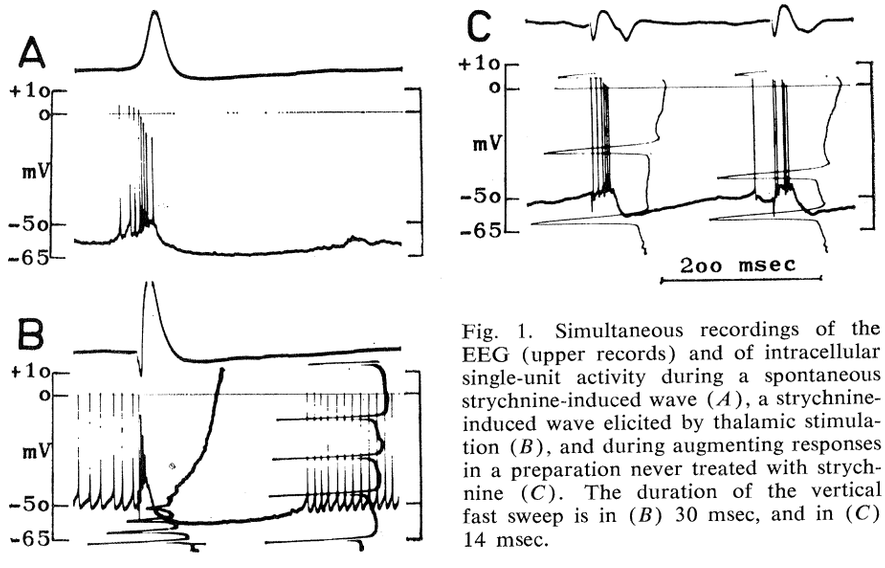

Fig. 1. Simultaneous recordings of the EEG (upper records) and of intracellular single-unit activity during a spontaneous strychnine-induced wave (*A*), a strychnine-induced wave elicited by thalamic stimulation (*B*), and during augmenting responses in a preparation never treated with strychnine (*C*). The duration of the vertical fast sweep is in (*B*) 30 msec, and in (*C*) 14 msec.

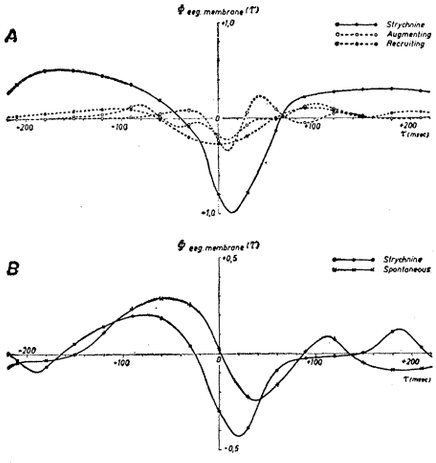

Fig. 2. Samples of cross-correlograms of evoked activities (*A*), and of normal (spontaneous) and strychnine-induced activities (*B*). Delay increments: in (*A*), 2 msec; in (*B*), 10 msec. Integration time: 0.4 second (*A*), 2.0 seconds (*B*). Calibration of ordinates: (*A*) highest strychnine-induced value of correlogram normalized to −10; (*B*) time-series correlation coefficient.

mechanism of strychnine seizures in the cortex now no longer seems to be the same as in the motoneuron where it is mediated by way of blockage of inhibitory postsynaptic potentials (Fig. 1) (*12*).

In Fig. 2*B*, average types of correlograms of spontaneous activities are shown. In this figure the ordinate scale represents the actual time series correlation coefficient $\rho_{eeg,membrane}$ (τ). The shape of all these correlograms depends especially upon the configuration of EEG waves which are predominantly monophasic-negative in the patterns for strychnine poisoning and recruiting. The EEG waves are accompanied at the single unit level by depolarizing waves of the membrane potential, the result being a negative correlation coefficient between both signals. During augmenting and spontaneous normal activity, biphasic EEG sequences occur which are also reflected in the correlograms. Very often a phase lead of one or the other of these signals can be observed on those correlograms which do not have their negative maximum at $\tau = 0$.

Table 2 summarizes the result of 40 correlograms. We measured this phase lead by different methods. If one assumes that this lead is probably due to electrotonically propagated transients between the two recording electrodes having a distance of 2 to 3 mm, then velocities in the order of magnitude of 10^{-1} m sec^{-1} can be calculated. Such velocities have been reported for slowly conducted dendritic potentials (*15*) and for slow waves which were ascribed to be dendritic processes (see *16*).

We found no differences between the correlograms of identified pyramidal tract cells and non-identified cells. But it is impossible to draw any conclusions about the extension of the dendritic ramifications and about the size of those cells that do not respond to antidromic stimulation (recurrent inhibition, anodal polarization), or to spot routinely the cells from which the recordings were obtained by any histological method. In addition, the time constants of synchronous events in different phases of membrane transients and EEG waves have been determined by graphical methods which show clearly that the time constants of both signals are comparable (Table 1) (*17*).

Evidently, there are close relationships between EEG waves and slow membrane transients which can be expressed quantitatively both by time-series analysis and by determining the time constants of both signals. Our findings supported at first the hypothesis that the EEG is a summation of postsynaptic potentials. But recent findings have also shown that there is no fundamental difference between graded dendritic responses and excitatory postsynaptic potentials elicited at axo-dendritic synapses (*10, 18*). In latency and firing level, these dendritic excitatory postsynaptic potentials exhibit differences from supposed axo-somatic excitatory postsynaptic potentials, and also their amplitude is not increased by hyperpolarizing currents injected through the microelectrode in the cell body.

Because of (i) some characteristics of dendritic elements in the conduction velocity of EEG phenomena, and because of (ii) the possibility that postsynaptic potentials are generated at more distal parts of dendrites, especially during spindle activity of the EEG, we feel justified in interpreting both EEG waves and slow membrane transients as being potential changes generated at the membranes of the cell bodies and of the dendritic ramifications of the cortical neurons.

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