EFFECT OF STRUCTURE ON FUNCTION IN MODEL NERVE NETS

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ABSTRACT A theoretical analysis has been made on the effect of the pattern of interneuronal connectivity in model nerve nets on the activity of these nets. Two types of nets have been investigated: one in which the likelihood of a connection between a given neuron and any other element in the net is given by a Poisson probability distribution, and a second type in which the pattern of interconnection follows a Gaussian distribution. An analytical treatment is presented of the equations for noiseless nets in these two conditions. The principal result is that nets with Poisson connectivity law are activated by extraneous firing of a single neuron and continue in spontaneous activity indefinitely. On the other hand, similar nets in which the connections are, however, distributed according to a normal connectivity law, exhibit a definite threshold and produce spontaneous activity only subsequent to extraneous activation of a substantial fraction of the population. Moreover, spontaneous activity in Gaussian nets, but not in Poisson nets, becomes extinguished if the number of active neurons falls below the critical threshold. Some neuroanatomical implications are discussed which suggest that the pyramidal system of the cerebral cortex and other neuronal systems histologically characterized by large numbers of synapses per neuron may incorporate a Gaussian connectivity law, whereas a Poisson law may be characteristic of these cortical layers and nuclei primarily containing granule cells.

INTRODUCTION

Phylogenesis of the central nervous system from amphibia to mammals has been marked by the appearance of new brain structures as well as structural modification and specialization of archaic structures. The emergence in mammals of a neocortex characterized by a multilayered nerve cell arrangement, combined with the retention in the hippocampus of essentially monolayered paleocortex, constitutes an outstanding example of this evolutionary process. More detailed scrutiny reveals additional structural specializations; thus, sensory neocortex is five-layered, whereas the motor cortex shows much less distinct stratification. On the other hand, thalamic nuclei, with the exception of the lateral geniculate, do not show any clear laminar

organization. The question naturally arises whether these structural features may reflect, and perhaps determine, fundamental differences in the mode of operation of distinct brain structures. Alternatively, the possibility may exist that such structural specializations merely represent anatomical "accidents of development," perhaps reflecting phylogenetic origins, but playing a functional role no more significant than, for example, the appendix or the coccygeal vertebrae in man.

It is difficult to provide an answer to this question from the presently available anatomical and physiological data. Although substantial neurohistological data on one hand and neurophysiological information on the other hand are available, meaningful correlation of these two sets of data can only be accomplished in very isolated instances. In general, unlike recording from invertebrates, where the simplicity and viability of the nervous system make it feasible to observe the elements recorded from, physiological studies of the mammalian central nervous system (CNS) are performed in a "blind" fashion and it is exceedingly difficult to correlate these studies with the microscopical anatomy of the tissue.

While the ultimate answer to the question of functional meaning of anatomical structure eventually must come from increased sophistication in experimental design and methodology, some insight may be obtained through the use of models. It is, of course, impractical to create a model which is a perfect replica of the system under study; to do so would presuppose perfect knowledge of the system—obviating the need for the model. Rather, one may choose a certain smaller subset of properties and employ the model to study the effect of these properties on operation of the model.

In the present study, we investigate the relationship between structure, as expressed in pattern of interneuronal synaptic connectivity, and "spontaneous" activity in finite nerve-cell assemblies. In constructing models of such neuron assemblies, connectivity among individual elements may be specified to follow a given probability law, maintaining all other parameters constant. It will be shown that the probability law selected to specify the connectivity pattern has a profound effect on subsequent activity in the system.

METHODS

In previous studies using probabilistic neural nets (Harth et al., 1969; Anninos et al., 1970), the following nomenclature has been adopted:

A	Total number of neurons in the net.
τ	Synaptic delay.
α_n	The fraction of active neurons at $t = n\tau$.
h	The fraction of inhibitory neurons.
μ^+	The average number of outgoing connections per excitatory neuron.
μ^-	The average number of outgoing connections per inhibitory neuron.
K^+	The average coupling coefficient for excitatory neuron, being a measure of synaptic efficacy in producing a potential change in the postsynaptic neuron.

K^- The average coupling coefficient for inhibitory neuron.

$\eta = \mu(\theta/K^+)$ Minimum number of EPSP necessary to trigger a neuron in the absence of inhibitory inputs.

$\eta' = \mu\left(\frac{\theta - mK^-}{K^+}\right)$ Minimum number of excitatory inputs necessary to trigger a neuron which has received m inhibitory inputs.

The elementary unit in all neural nets is the neuron. Each neuron is in one of two possible states: resting or active. The transition from the resting to the active state occurs when the sum of the postsynaptic potentials (PSP) arriving at the cell exceeds a certain value called threshold θ. PSP may be excitatory (EPSP) or inhibitory (IPSP), producing changes in membrane potential which take the cell closer to or further away from the firing threshold. A neuron may be either excitatory or inhibitory. In the first case, all of its axon branches will generate EPSP; in the second case, they will generate IPSP. If a neuron fires at time t, it produces the appropriate PSP after a fixed time interval t, the synaptic delay. All PSP arriving at a neuron are summed instantly and, if they exceed the threshold, will cause the neuron to fire without further delay. After firing the neuron is insensitive to further stimulation for a period called the refractory period. Postsynaptic potentials, if below threshold, will persist with or without decrement for a period of time, called the summation time. For our purposes it will be assumed that the refractory period is greater than the synaptic delay, but less than twice the synaptic delay. This assumption leads to the following effect: if a number of neurons are fired synchronously at time t, then all neural activity resulting from this initial activity will be restricted to times $t + \tau$, $t + 2\tau$, etc.

Of course, the assumption that PSP duration does not exceed the absolute refractory period is not realistic in the majority of physiological preparations known (cf. e.g., Elul, 1972). However, this admittedly arbitrary constraint greatly simplifies the formal representation; it can also be shown that the behavior of the artificial nets considered here is fundamentally similar for refractory periods shorter and longer than PSP duration, although higher-order difference equations are required to describe net activity in the latter case (Wong and Harth, 1973).

Another assumption implicit in the present treatment is that of noiselessness. Again, it is likely that physiological systems exhibit some degree of noise (e.g., Levitan et al., 1968), although it is likely that the importance of noise decreases as the dimensions of the neuron increase. The possible effect of noise on the behavior of the nets studied here has been investigated in considerable detail (Anninos, 1969; Anninos et al., 1970). Essentially, the introduction of noise results in some increase in the percentage of active neurons. This increase results in a shift in the dynamic properties of the net, as characterized in the following section, but so long that the noise is not overwhelming, does not alter the overall behavior of the net with regard to the presence or absence of sustained activity which is determined by the dynamic properties (see Appendix). Thus, the assumption of noiselessness will not materially affect the analysis that follows.

From the assumptions stated above, a general expression for the expectation value $\langle \alpha_{n+1} \rangle$ of the activity at $t = (n + 1)\tau$ can be written as (Anninos et al., 1970)

$$\langle \alpha_{+1} \rangle = (1 - \alpha_n) P(\theta, \alpha_n), \tag{1}$$

where $P(\theta, \alpha_n)$ is the probability that the excitation on a given neuron exceeds the firing threshold θ. As shown in previous work (Anninos et al., 1970), $P(\theta, \alpha_n)$ can be expressed, depending on the validity of approximations, in terms of binomial or Poisson distributions of the number of excitatory and inhibitory inputs to a cell. In previous work with compound

nets it was also noted that the Poisson distribution may be replaced by a Gaussian distribution in order to simplify Eq. 1 without going into the physical and physiological significance of this substitution.

In the present paper we examine the significance and consequences of this replacement of the Poisson probability distribution by a normal one. We first proceed to develop an explicit derivation of Eq. 1 for a normal probability distribution.

THEORETICAL ANALYSIS AND RESULTS

If the average number of active inputs per neuron becomes large, de Moivre's approximation may be used and the distribution becomes Gaussian. Because of a well-known theorem which states that the sum of any number of independent Gaussian distributions also is Gaussian, we may make the following simplifications (Anninos et al., 1970): Let the total PSP input to a neuron at time $t = (n + 1)\tau$ be given by

$$e_{n+1} = lK^+ + mK^-, \tag{2}$$

where l and m are the number of EPSP and IPSP, respectively. If both l and m are large, their distributions may be approximated by normal distributions about their respective average values $\bar{l} = \alpha_n\mu^+(1 - h)$ and $\bar{m} = \alpha_n\mu^- h$. The distribution of e_{n+1} therefore is also normal, since its variance is the sum of the variances of l and m and the probabilities for l and m are independent of one another. The average PSP will be given by

$$e_{n+1} = \alpha_n[\mu^+(1 - h)K^+ + \mu^- hK^-]. \tag{3}$$

The variance of e_{n+1}, say $\delta^2{}_{n+1}$, is then given by

$$\delta^2{}_{n+1} = \alpha_n[\mu^+(1 - h)(K^+)^2 + \mu^- h(K^-)^2]. \tag{4}$$

The probability $P(\theta, \alpha_n)$ that the PSP exceeds a threshold θ now becomes

$$P(\theta, \alpha_n) = \frac{1}{\sqrt{2\pi}} \int_{x_{n+1}}^{\infty} \exp(-x^2/2)\, dx, \tag{5}$$

where

$$x_{n+1} = (\theta - \bar{e}_{n+1})/\delta_{n+1}.$$

Eq. 5, when used in conjunction with Eq. 1, gives values for $\langle\alpha_{n+1}\rangle$ which are in fair agreement with the results of computer simulation even for values of μ^+, μ^-, as low as 10.[1]

Let us proceed to our original goal, i.e., to see the significance of the replacement

[1] ANNINOS, P. A., and R. ELUL. 1973. In preparation.

of Poisson probability distribution by a normal one. Using our previous assumption, Eq. 1 takes the form:

$$f(x) = (1 - \alpha_n)P(x). \tag{6}$$

In order to investigate whether there is any difference in behavior of the curve α_n vs. α_{n+1} for Poisson and normal distributions, we examine the slope of $f(x)$ at the origin. To accomplish this, we investigate the partial derivative of $f(x)$ with respect to α_n for $\alpha_n = 0$:

$$\left.\frac{\partial f(x)}{\partial \alpha_n}\right|_{\alpha_n=0} = \frac{\partial}{\partial \alpha_n}(1 - \alpha_n)P(x)\,|_{\alpha_n=0}. \tag{7}$$

From Eq. 7 it follows that:

$$\left.\frac{\partial f(x)}{\partial \alpha_n}\right|_{\alpha_n=0} = -P(x) + (1 - \alpha_n)\frac{dP(x)}{dx}\left.\frac{dx}{d\alpha_n}\right|_{\alpha_n=0}. \tag{8}$$

Using (5) we get:

$$\frac{dP(x)}{dx} = -\frac{1}{\sqrt{2\pi}}e^{-\frac{x^2}{2}} \tag{9}$$

if we assume $|K^+| = |K^-| = 1$ and $\eta = \theta/K^+$ it follows that

$$X = \frac{\theta - \bar{e}}{\delta} = \frac{\eta - \alpha_n\mu^+(1 - h) + \alpha_n\mu^- h}{\sqrt{\alpha_n\mu^+(1 - h) + \alpha_n\mu^- h}}. \tag{10}$$

Let us introduce $\mu^+(1 - h) = q$ and $\mu^- h = p$, then Eq. 10 takes the following form:

$$X = \frac{\eta - \alpha_n q + \alpha_n p}{\sqrt{\alpha_n(p + q)}}. \tag{11}$$

If we differentiate Eq. 11 with respect to α_n we obtain:

$$\frac{dx}{d\alpha_n} = \frac{-\eta + \alpha_n p - \alpha_n q}{2\alpha_n\sqrt{\alpha_n(p + q)}}. \tag{12}$$

Therefore Eq. 8 takes the form:

$$\begin{aligned}\left.\frac{\partial f(x)}{\partial \alpha_n}\right|_{\alpha_n=0} &= -P(x) - \frac{(1 - \alpha_n)}{\sqrt{2\pi}}\exp(-x^2/2)\left.\frac{-\eta + \alpha_n p - \alpha_n q}{2\alpha_n\sqrt{\alpha_n(p + q)}}\right|_{\alpha_n=0} \\ &= -P(x) - \frac{1 - \alpha_n}{\sqrt{2\pi}}\exp(-x^2/2)\frac{x}{2\alpha_n}\end{aligned} \tag{13}$$

$$+ \frac{2(1 - \alpha_n)}{\sqrt{2\pi}} \exp(-x^2/2) \frac{\eta}{2\alpha_n \sqrt{\alpha_n(p + q)}} \Big|_{\alpha_n \to 0}.$$

Using l'Hospital's rule and noticing that for $\alpha_n \to 0$ the $X \to \infty$, it can be easily seen that

$$\lim_{\alpha_n \to 0} \frac{\partial f(x)}{\partial \alpha_n} \to 0 \qquad (14)$$

In other words, the slope of $f(x)$ at the origin tends to zero for $P(\theta, \alpha_n)$ which follows a Gaussian distribution. Since $f(x) = \langle \alpha_{n+1} \rangle$, it follows that in a net with Gaussian probability distribution of discharge, the expectation of α_{n+1} approaches zero for low values of α_n (Fig. 1 *B*). In contrast, a Poisson net with the same ratio of excitatory and inhibitory elements and with the same threshold shows a very different behavior at the origin, as also shown in Fig. 1 *B*, even though for larger

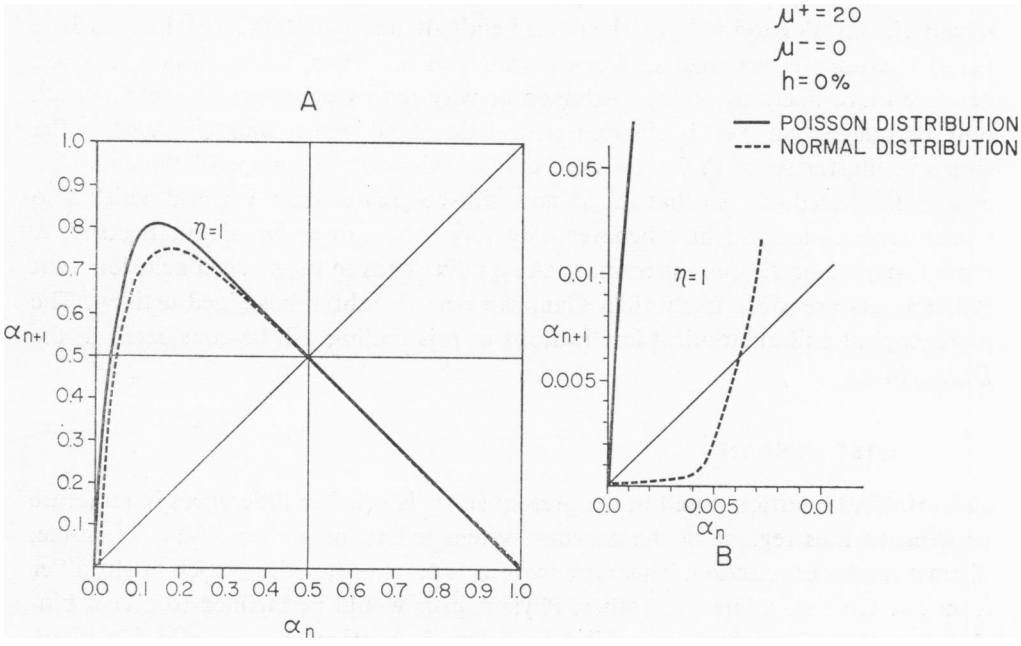

FIGURE 1 (*A*) Effect of the pattern of connectivity on dynamical behavior of the net. The two curves describe activity of the same net, except that in one instance connections are made according to a Poisson law (solid line), while in the second instance a normal distribution is used (interrupted line). (*B*) Magnified plot at the origin reveals that the net with Poisson distribution is above the 45° diagonal at the origin and consequently will respond in sustained activity to extraneous activation of a single cell. The same net, when organized following a normal distribution, lies under the 45° diagonal at the origin and will exhibit sustained activity only if more than 0.6% of the population are activated simultaneously (α_n = 0.006 at the intersection with the 45° diagonal).

percentages of firing neurons α_n the Gaussian and Poisson nets exhibit rather similar behavior (Fig. 1 *A*). This difference at the origin is the critical result of the present study. The behavior of nerve nets when only a very low number of neurons is firing quite obviously would determine whether a modest extraneous input can excite activity in the net. Equally important, should the activity at the net in the course of time fall to a low level, again the behavior characteristic with low α_n would determine whether activity in the net will die out altogether or whether it may continue. Thus, the relationship at the origin between α_n and α_{n+1} determines whether the net is or is not capable of sustained activity. For nets of the type considered here, the implications on dynamic behavior of the ratio α_n/α_{n+1} close to the origin have been analyzed in detail by Harth et al. (1969), Anninos (1969), and Anninos et al., (1970). A brief outline of this analysis is given in the Appendix. However, already from inspection of Fig. 1 *B* it is clear that in a Gaussian net α_{n+1} would tend to be smaller than α_n, so that whenever α_n is small (either in a previously quiescent net which has just received minimal extraneous input, or in a previously active net which has minimal activity at this point), α_{n+1} is likely to be even smaller so that activity of the net would die out. Poisson nets, on the other hand, are immune to this risk inasmuch as $\langle\alpha_{n+1}\rangle$ is always larger than α_n for low values of α_n. Thus, in conclusion, Poisson nets are more likely to exhibit sustained activity than Gaussian nets which would only become active if α_n is above a critical threshold (approximately 0.004 in the situation illustrated in Fig. 1 *B*—note that at this value the slope of the curve increases to exceed 45°, so that $\langle\alpha_{n+1}\rangle$ now will be greater than α_n), and which also would come to a standstill whenever only very few neurons fire simultaneously. A more formal analysis, presented in the Appendix, leads to the same conclusion, that Poisson nets are more likely than Gaussian nets to exhibit sustained activity. The physiological and anatomical implications of this finding will be considered in the Discussion.

DISCUSSION

The principal question posed in the present study is whether differences in structure between various regions of the nervous system reflect, or perhaps even determine, distinct modes of function. There are various aspects of function which might differ from one CNS structure to another. Physiologists would be inclined to accept bioelectric activity in general, and spike discharges in particular, as a valid, if limited, estimator of nervous function. Thus the present study up to this point concentrated on analysis of the dynamic pattern of spike firing as represented in Eq. 1, i.e., $\langle\alpha_{n+1}\rangle = (1 - \alpha_n)P(\theta, \alpha_n)$ which describes the relationship between the level of activity, expressed in terms of the number of discharging neurons at a given period α_{n+1}, and the activity in the immediately preceding period α_n. Explicit solution for this equation has been derived in the Results section of the present paper for the case where $P(\theta, \alpha_n)$ is a normal variable. The corresponding solution for the Poisson case has

been studied in detail in earlier work (Csermely, 1968; Harth et al., 1969; Anninos et al., 1970), which is briefly summarized in the Appendix. Comparison of these two sets of results shows that, given two nets with an identical proportion of excitatory and inhibitory synapses as well as firing threshold, but differing in the function $P(\theta, \alpha_n)$, the resultant activity in the two cases is quite different, the Poisson net being capable of sustained activity under conditions in which activity in Gaussian nets would become quenched very rapidly (Fig. 1).

The question which must be dealt with at this point is that of the interpretation of the function $P(\theta, \alpha_n)$ in real nerve nets in the CNS: do different $P(\theta, \alpha_n)$ entail differences in structure of these nets, and if so, what are these differences?

To answer this question, we need to consider Eq. 1 in somewhat greater detail. This equation states that the number of firing neurons (as percentage of the total neuron population) in a given cycle is determined by the number of neurons available for firing during that particular cycle ($1 - \alpha_n$ is the fraction of available neurons, since those α_n neurons which fired in the preceding cycle are refractory during the current cycle). Eq. 1 also states, however, that only a fraction of the available population will fire, and that this fraction is determined by $P(\theta, \alpha_n)$. Considering that the threshold is fixed in this model, and indeed also physiologically can be taken as a stable factor at least over short periods of time, the only variable which determines $P(\theta, \alpha_n)$ is the number of neurons firing in the preceding cycle, α_n. According to whether a Gaussian or Poisson probability function is selected, $P(\theta, \alpha_n)$ will either be a Gaussian or Poisson variable determined by α_n. Specifically, the value of $P(\theta, \alpha_n)$ in successive cycles would be distributed according to either normal, or Poisson distribution, with the independent variable being the number of neurons firing in the preceding cycle α_n.

It is appropriate to inquire how this functional dependence can be satisfied in "real" neuronal nets, i.e. in nets where the pattern of interneuronal connections is inalterable. One way to satisfy this relation is if the number of synaptic connections received by each cell would vary as a Gaussian variable, or alternatively as a Poisson variable. Based on the assumption that synaptic potentials sum algebraically—an assumption which is widely accepted in regard to the analysis of physiological data (see, however, Elul and Adey, 1966 for further discussion)—it is clear that different cells in the net would have a different probability of discharging following the discharge of, say, 10% of the cells in the immediately preceding cycle. Moreover, on the assumption of algebraic summation of synaptic inputs, the firing probabilities of these cells, being directly related to the number of afferent connections, will be distributed following a Poisson, or Gaussian probability distribution, respectively. In this way, Eq. 1 will be satisfied in nerve nets in which the number of connections received by each cell (and in a closed system—also originating in each cell) represents a Poisson, or alternatively a Gaussian variable. Our results may therefore be restated as follows: A neuronal net where the *number* of connections received by each neuron is distributed according to a Poisson function will exhibit sustained activity.

On the other hand, a net where the number of connections reaching each particular nerve cell varies according to a normal distribution, will not be capable of sustained activity.

These results are of significant interest in pointing to a hitherto unconsidered parameter, which may be worthy of examination in histological material: the probability distribution of the number of synapses received by individual cells in a given nucleus or region of the brain. Although there are a number of studies in the literature dealing with quantitative aspects of synapse populations, the variance and the mean of counts in individual neurons have not been documented in most of these studies, and the probability function of distribution of these connections has not been investigated.

Notwithstanding the absence of specific information on the probability distribution of the numbers of synaptic inputs to individual neurons in different brain structures, we may proceed in our investigation of the present results in a more general fashion, given certain well-known properties of the normal distribution. It is important to recall in this context that, for a large number of events (i.e. in our model, a large number of interconnections), the Poisson distribution converges toward the normal distribution (cf. e.g., Feller, 1957, p. 176–178; Cox and Lewis, 1966, Chap. I). Thus Poisson neuronal nets may be viewed as approximately Gaussian whenever the number of synaptic connections is relatively large. Insofar as the normal distribution is the limiting distribution for great many experimental distributions when the number of elements becomes large, it is also likely that in general, most systems with large numbers of synaptic connections per nerve cell would tend toward the Gaussian case discussed in the present study. For these reasons, it appears that the conclusions reached here regarding nets characterized by Gaussian probability law, may be applicable within the limitations of the basic assumptions of the model, and with only minor modification, to most real neuronal centers for which there is histological evidence of rich systems of interconnections. Indeed, these results would equally apply also to nerve nets with Poisson distribution of the interconnections, provided only that the average number of connections (the Poisson parameter λ) is above 50 (cf. e.g., Cox and Lewis, 1966, p. 21). Thus, our results may be further generalized to suggest that the average number of interneuronal connections in a given CNS structure may be the significant parameter in determining spontaneous activity: all structures having large numbers of interconnections per nerve cell would be unlikely to exhibit intrinsic sustained activity. Such spontaneous activity appears from our results to be more likely to originate in neuronal nets with small average number of synapses per neuron.

At first glance, this conclusion might appear rather unexpected. However, some intuitive insight to the underlying neuronal mechanisms, as well as to the assumptions implied in our treatment of this problem, may be gained from consideration of the chain of events leading to neuronal discharge. If a nerve cell is characterized by a given firing threshold which, when exceeded, results in spike discharge, one may

then contrast two anatomical situations: one in which there are only few synaptic contacts reaching the cell in question, and a second in which the cell receives a large number of synaptic inputs. If in either case firing is dependent on simultaneous excitation of a certain *percentage* of the total synaptic input (assuming that the ratio of excitatory and inhibitory synapses is the same in both situations so that the role of the inhibitory inputs may be disregarded for the moment), then it is clear that firing in neurons with large numbers of synaptic inputs would require synchronized activation of a substantial number of synapses; whereas in neurons with few synapses, firing may ensue even from activation of a single excitatory synapse. Thus, the system where neurons receive small numbers of synaptic connections is likely to exhibit a less "controlled" pattern of activity—and indeed also "spontaneous" discharges.

It may be argued that this reasoning presupposes that the threshold for firing is the same in systems with small and large numbers of synapses, and consequently plays down the importance of any single synaptic input to cells receiving large numbers of synapses. However, studies on nerve cells in the cerebral cortex, which probably receive tha largest number of synapses per cell in the CNS, invariably reveal firing rates of less than 20/s, indicating that the threshold for firing is not lower in these cells than in peripheral neurons with much lower number of synapses per cell (which, in fact, often fire at rate around 100/s). Indeed, it is conceivable that effector cells in the CNS, such as the cortical pyramids and the cerebellar Purkinje cells, have evolved to receive large number of inputs per cell precisely in order to reduce the probability of "spontaneous" (i.e., unexpected) firing which may affect peripheral muscular posture and movement. In contrast, granule cells and Golgi type II cells which typically have much lower count of synaptic inputs typically act as interneurons, so that any "spontaneous" activity originating in them would be buffered by the multisynaptic pyramidal cells on which they converge. It is interesting to note that slabs of cortical tissue, when isolated from thalamic and other inputs, do not exhibit spontaneous activity (Burns, 1950, 1951), perhaps reflecting the large average number of synapses impinging on each neuron in this tissue, which number in several thousands (e.g., Kositzyn, 1964).

APPENDIX

The neural nets considered in this paper are finite and self-contained. In such nets, without temporal summation of excitation and inhibition, and with refractoriness extending only one time interval, the number of "cells" active at any instant is entirely determined by the number of cells active in the preceding stage. In this sense, the probability of activity in the net represents a Markoff chain, where the fraction of the population which may be activated at any time $n + 1$ is $(1 - \alpha_n)$, α_n being the number of cells active in the preceding time interval n. As seen from Eq. 1, the expectation of active cells must be $\langle \alpha_{n+1} \rangle \leq (1 - \alpha_n)$ since $P(\theta, \alpha_n)$ cannot exceed unity. The functional interpretation of $P(\theta, \alpha_n)$ has been considered in the discussion. Here we are concerned with only dynamics of the net. Since dynamically the net may be represented as a Markoff chain, we need only consider the relation-

ship $\langle \alpha_{n+1} \rangle / \alpha_n$ (Fig. 2). Clearly, if $\alpha_n = 1$, α_{n+1} must be zero, as *all* elements will be refractory at time τ_{n+1}. In general, dynamic behavior of a given net can be expressed in terms of the curve $\langle \alpha_{n+1} \rangle / a_n$. A detailed description of these curves has been given (Harth et al., 1969; Anninos, 1969; Anninos et al., 1970). Here only a few relevant points will be emphasized. Whenever this curve crosses the 45° diagonal, clearly $\langle \alpha_{n+1} \rangle = \alpha_n$. Below the 45° diagonal, $\langle \alpha_{n+1} \rangle < \alpha_n$, and unless the curve changes its slope, activity in the net will gradually decay.

Conversely, if the curve lies above the 45° diagonal, the number of active cells will increase. This property is of particular importance when the *initiation* of activity in the net is considered. Inasmuch as the nets considered here are noiseless, it is clear that at the very least, *one* "cell" must be fired extraneously in order to obtain any activity at all. We are therefore concerned with events around the origin of the curves in Fig. 2. From the preceding discussion it is clear that if the curve at the origin lies above the 45° diagonal, additional cells would be recruited to discharge; on the other hand, the extraneous input cannot be successful in exciting sustained activity in the net if the curve $\langle a_{n+1} \rangle / \alpha_n$ is below the 45° diagonal. An additional situation also needs to be considered, where the curve initially lies under the 45° diagonal, but subsequently, for larger values of α_n, it exceeds this diagonal. In this case, the net will exhibit sustained activity if, and only if, the input is large enough to reach this point (Harth et al., 1969).

These considerations served as basis for the classification of artificial nets of this type into three classes by Anninos et al. (1970). The three types of nets are shown in Fig. 2: class A, which for low values of α_n always is above the 45° diagonal, and consequently exhibits sustained activity. (It should be noted that, although the curve predicts very low values of

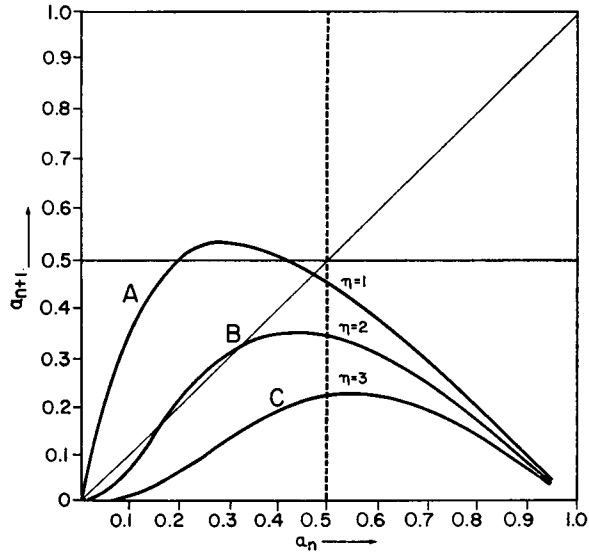

FIGURE 2 The three types of model nerve nets, classified in terms of their capability of generating sustained activity following extraneous excitation of even a single element (*A*); only if a certain threshold is exceeded (*B*); or not at all (*C*). The expectation of number of firing neurons α_{n+1} plotted as a function of the number of neurons discharging in the preceding time interval α_n. All nets have the same parameters, except that the number of EPSP necessary to trigger a neuron differs from *A* to *C*.

α_{n+1} for large α_{n+1} (e.g. 0.9), the low α_{n+1} in next time interval represents low α_n; in other words, the net returns to the region above the 45° diagonal.) Class B, as seen in Fig. 2, will exhibit sustained activity only if the initial extraneous input exceeds a certain "threshold." For this reason, its activity is liable to become extinguished if it happens to exceed a certain level (0.8 in Fig. 2). Thus, activity in class B net is likely to be relatively short-lived. Nets of class C are totally incapable of sustained activity since they never reach the 45° diagonal for any given initial activity.

We are now in position to evaluate the results of the present study. Fig. 1 has shown in solid line, the relationship $\langle\alpha_{n+1}\rangle/\alpha_n$ for nets where a Poisson probability function is used. It can be seen that these belong to class A., i.e., following activation of even a single element by an extraneous input, these nets will exhibit sustained activity indefinitely. Using the same parameters, but employing a normal distribution, it is evident that the curve $\langle\alpha_{n+1}\rangle/\alpha_n$ near the origin (see inset) is below the 45° diagonal, and thus represents a class B net, which will maintain activity only when it exceeds a certain threshold at which the curve crosses the diagonal.

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