

A Mathematical Model for the Decay of Short-term Memory with Age

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A mathematical neural net model based on our previous studies (Anninos *et al.*, 1970; Anninos, 1972) is proposed here to show that the short-term memory of events decays with man age. In particular, in this work we try to explain why recent memories die out with age before the establishment of permanent memory. As it was shown we lose some connections due to the loss of a large number of neurons with age, and in our model this corresponds to a smaller hysteresis loop which, according to our assumption, represents the short-term memory of an event. Thus we showed that if we decrease the number of connections even further the hysteresis loop will vanish to a single curve which, according to Katchalsky & Oplatka (1969), corresponds to a memory-less system.

1. Introduction

In previous studies (Anninos *et al.*, 1970) we constructed a neural net model consisting of discrete sets of neurons referred to as “netlets”. Neuronal activity within a netlet was considered to be endogenous or triggered by afferent fibers. It was also possible to be transient or self-sustained. The netlets were assumed to be coupled to one another in a way which was described as randomness-in-the-small and structure-in-the-large. The second assumption was concerned with the appropriate description of neural dynamics. The statement was made (Harth *et al.*, 1970) that the spatial and temporal microstructure of activity may be disregarded. The dynamical variables considered significant in that model were the levels of activity, i.e. the fractional number of neurons firing in each netlet. With these sets of assumptions, the dynamics of some simple probabilistic neural nets were studied. Of particular interest were the conditions under which a netlet will go into sustained activity by afferent excitatory or inhibitory inputs. Hysteresis effects were common under these conditions and it was assumed that such effects may represent a type of short-term memory (Harth *et al.*, 1970; Anninos, 1969, 1972; Anninos *et al.*, 1970; Wilson & Cowan, 1972).

In the present study an explanation of the reasons for which the short-term memory of a past experience, represented by a hysteresis loop, will fade away with age is attempted. In particular, we explain why this happens before the establishment of permanent memories.

2. Methods

(A) UNIT PROPERTIES

The present investigation makes use of the neural net model developed in our previous work (Anninos, 1969, 1972; Anninos *et al.*, 1970). It was assumed that if a neuron fires at time t , it produces the appropriate PSPs (post-synaptic potentials) after a fixed time interval called synaptic delay τ . All PSPs arriving at a neuron are summed instantly and, if they exceed the specified threshold, will cause the neuron to fire. After firing neurons are insensitive to further stimulation for a period of time called the "refractory period". For our purposes we have assumed that the refractory period is greater than the synaptic delay but less than twice the synaptic delay. Temporal summation occurs without decrement, but only for periods less than one synaptic delay. Following the absolute refractory period, the threshold of the neuron is immediately restored to normal. Thus a neuron which fires at $t = n\tau$, will be insensitive at $t = (n + 1)\tau$ and fully recovered at $t = (n + 2)\tau$. The PSPs on a particular neuron at $t = n\tau$ depend only on the firing record of the netlet at $t = (n - 1)\tau$. Clearly, therefore, under the present set of assumptions, the dynamics of the netlet is a Markov process. We defined the activity a_n of a netlet as the fractional number of neurons firing at $t = n\tau$. The activity is a scalar and does not specify which neurons in the netlet were firing.

(B) PARAMETERS OF NEURAL NET MODEL

- τ Synaptic delay.
- A Total number of neurons in the netlet.
- h Fraction of inhibitory neurons in the netlet.
- μ^+ The average number of axon branches emanating from an excitatory neuron.
- μ^- The average number of axon branches emanating from an inhibitory neuron.
- K^+ The average excitatory post synaptic potential (EPSP) produced by an excitatory neuron in arbitrary units.
- K^- The average inhibitory post synaptic potential (IPSP) produced by an inhibitory neuron in arbitrary units.

- θ Firing threshold of the neurons in the netlet.
- η The minimum number of EPSPs necessary to trigger a neuron in the absence of inhibitory inputs.
- η' The minimum number of EPSPs necessary to trigger a neuron in the presence of inhibitory inputs.
- α_n The activity, i.e. the fraction of active neurons in a netlet at $t = n\tau$.
- σ^+ The fraction of active fibers, i.e. those carrying action potential (AP) at a particular instant.
- μ_0^\pm The afferent parameters.
- A_0 The total number of afferent fibers.

(C) EXPECTATION VALUE OF THE ACTIVITY WITH SUSTAINED INPUTS

Let us consider a cable of afferent fibers attached to an isolated neural net. Upon entering the netlet, each of these fibers splits and makes synaptic connections with μ_0^+ neurons if it comes from an excitatory neuron, or μ_0^- neurons if it comes from an inhibitory neuron. Each fiber branch is assumed to make one synaptic connection with a neuron in the netlet. The neurons are selected randomly according to our assumption of local randomness. Let the total number of afferent fibers be A_0 . They may be considered to be axons of A_0 neurons in another network. Let K_0^+ be the average coupling coefficient of the synapses made by the afferent excitatory fibers. Further, let σ be the fraction of active fibers, i.e. those carrying action potentials at a particular instant. Assume that σ is either slowly changing or constant but that the number σA_0 of active fibers is chosen randomly at each time interval τ . The expectation value of the activity $\langle a_{n+1} \rangle$ for such steady inputs is calculated in a manner similar to that of the isolated netlets. Thus for the simple case with only excitatory neurons, the *a priori* probability that a neuron receives a total PSP exceeding its threshold is given by a function of the dynamical variables α_n and σ and the parameters $A_0, A, \mu^+, \theta, K^+, \mu_0^\pm, K_0^+$: (the μ_0^+ or μ_0^- refer to excitatory or inhibitory afferent fibers, respectively)

$$P(\alpha_n, \sigma) = e^{-\mu_0^\pm \sigma} \sum_{l'=0}^{l'_{max}} \frac{(\mu_0^\pm \sigma)^{l'}}{l'!} \left[1 - e^{-\alpha_n \mu^+} \sum_{l=0}^{l=n'-1} \frac{(\alpha_n \mu^+)^l}{l!} \right] \quad (1)$$

where

$$l'_{max} = \sigma \mu_0^\pm A_0$$

and

$$\eta' = u[(\theta \mp l'K_0^+)/K^+] \quad (3)$$

Here $u[x]$ is defined as the smallest integer which is greater or equal to x .

Because of the assumed refractoriness, $\langle \alpha_{n+1} \rangle$ is again given by

$$\langle \alpha_{n+1} \rangle = (1 - \alpha_n)P(\alpha_n, \sigma) \quad (4)$$

Equations (1)–(4) are special cases of the general equations developed in our previous studies (Anninos, 1969; Anninos *et al.*, 1970). In all data presented here we have assumed $A_0 = A$, and $K_0^+ = K^+/2$. When the intersections with the straight line $\langle \alpha_{n+1} \rangle = \alpha_n$ are plotted against σ we obtain what was called “phase diagrams” (Harth *et al.*, 1970; Anninos, 1969; Anninos *et al.*, 1970). The striking aspect of the phase diagram analysis of the previous work was the appearance of hysteresis loops. A slow change of the level of the afferent inputs was leading to irreversible changes in the steady state activity of the netlet. Such hysteresis effects in the form of high sustained activity in the netlet were assumed to provide a short-term memory of an event, according to the definition of Katchalsky & Oplatka (1966). In this model we have to take into account that with age we lose a large number of neurons (John, 1967). So, due to this fact

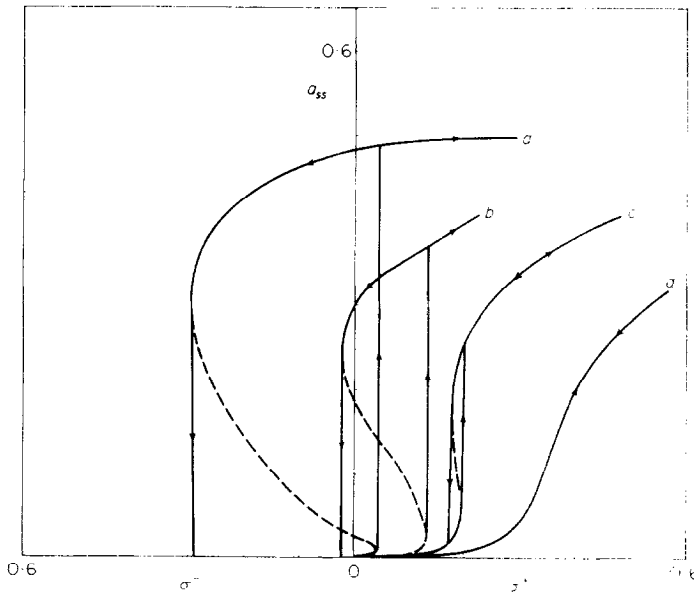


FIG. 1. Plot of steady state activities a_{ss} versus σ . These are phase diagrams showing regions of steady state activities (solid lines) and metastable activities (dotted lines) for sustained excitatory and inhibitory inputs σ^\pm and the corresponding hysteresis loops generated by slow changes of the σ s. For all curves the neural net parameters are, $A = A_0 = 1000$, $\eta = 2$, $h = 0\%$, $h_0 = 100\%$, and for curve (a) $\mu^+ = \mu_0^+ = 10$, (b) $\mu^+ = \mu_0^+ = 5$, (c) $\mu^+ = \mu_0^+ = 4$, and (d) $\mu^+ = \mu_0^+ = 3$.

the decrease of the number of neurons has as a consequence to lose some connections and consequently recent memories die out before the establishment of permanent memory. To explain this fact we use equation (4) for seven neural nets with parameters of high and low connectivity, which may correspond to young and old real biological brain, respectively. The results will be discussed in the next section.

3. Results

If we plot the steady-state values of activity obtained from equation (4) (by requiring $\langle \alpha_{n+1} \rangle = \alpha_n$) versus σ we obtain the phase diagrams and hysteresis loops which are shown in Figs 1 and 2. In Fig. 1 $A = A^0 = 1000$, $\eta = 2$, $h = 0\%$, $h_0 = 100\%$, and $\mu_0^\pm = \mu^\pm = 3, 4, 5$, and 10. In Fig. 2 $A = A^0 = 1000$, $\eta = 5$, $h = 0\%$, $h_0 = 100\%$, and $\mu_0^\pm = \mu^\pm = 5, 10$, and 30. For all nets $K^+ = 1$ and $K_0^\pm = 0.5$. As can be seen from Figs 1 and 2 by decreasing the number of connections (from 30 to 3) which is the case in the real brain with the age, the higher steady state of the memory item representation decreases and also the dotted line curve, which represents the threshold

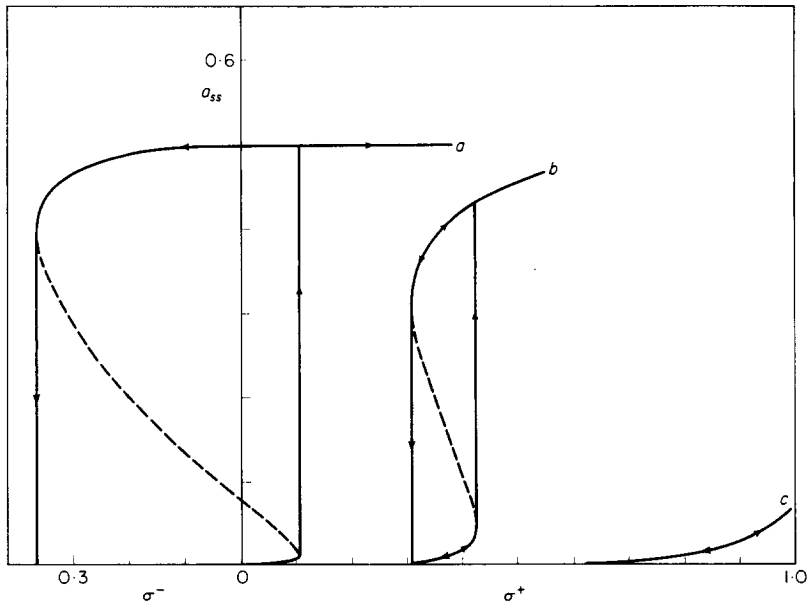


FIG. 2. Plot of steady state activities a_{ss} versus σ . These are phase diagrams, same as in Fig. 1, but here $A = A_0 = 1000$, $\eta = 5$, $h = 0\%$, $h_0 = 100\%$, and for curve (a) $\mu^+ = \mu_0^\pm = 30$, (b) $\mu^+ = \mu_0^\pm = 10$, and (c) $\mu^+ = \mu_0^\pm = 5$.

to trigger the net into higher sustained activity increases as expected. It is also clear from the same Figures that if the number of connections are further decreased this will result in a smaller hysteresis loop. This corresponds to suppression of short-term trace of recent items, and consequently it will be difficult to establish the permanent memory. This can be seen from curve d, which according to the definition of Katchalsky & Oplatka (1966) represents a memory-less system, as it consists of only equilibrium states.

4. Discussion and Conclusions

It has been known that when a stimulus pattern is presented to a subject, some kind of representation of this item becomes active in its visual cortex. This temporary representation is in the form of a reverberatory circuit or to use our term, in the form of hysteresis loops (Harth *et al.*, 1970; Anninos *et al.*, 1970; Anninos, 1972). The first step in the process of memorizing events in response to external stimulus seems to be a short-lived imprint (in our model the hysteresis loop). This is followed by a long-lived one if the pattern of the electrical activity in the brain is not disturbed during the critical interval for that to happen (Waugh & Norman, 1965). Since, as we mentioned above, in an old person we frequently lose some neurons (John, 1967) some pathways may be removed. Although as a whole the brain still functions, there will still be a recollection of past events, but the recent occurrences are the ones that can not be remembered due to the decrease of the higher steady state of the memory representation, and the increase of the threshold to trigger the net into higher sustained activity from the loss of the pathways with age.

Another strong evidence that supports our theory is the case of chronic alcoholics. The mammillary bodies (McCleary & Moore, 1965), small limbic structures in the hypothalamus, which are directly connected with the hippocampus are also important in memory processes in humans. Thus, chronic alcoholics suffering from long term nutritional deficiency frequently develop severe losses of recent memories. In these cases neuropathologists typically find damage in and around the mammillary bodies. This damage might cause the same result in humans as in the case with age, in which the loss of several neurons might develop losses of recent memories.

Thus, in conclusion, we might say that although our model is very simple regarding the present set of assumptions, (which lead to the dynamics of the netlets considered to be a Markov process), we were able to explain why recent memories in humans may not be recollected as they advance in age. Several more assumptions regarding refractoriness, summation time,

and relative refractory periods lead to non-Markovian dynamics, and such assumptions are presently under study to see how they will affect our current results.

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