MATHEMATICAL MODELS FOR NEURAL NETWORKS

VIOLET R. CANE
STATISTICAL LABORATORY, UNIVERSITY OF CAMBRIDGE

1. Introduction

The study of neural network models contributes indirectly to the study of the way in which brains work. The neurons which make up a brain have a number of known physiological properties; the brain operating as a whole has other known properties. By investigating the overall behavior of assemblies of cells which have some, but not all, of the properties of neurons and comparing this behavior with that of a brain it should be possible to decide the role and the relative importance of the properties that neurons have. In this paper examples of various models are given and others may be found from the references. Much of the recent work on networks has been influenced by ideas relating to the design of high speed digital computers; the question that is asked is, essentially, how a stimulus applied to an assembly at rest is transmitted or transformed by it. This is a static approach. A dynamic approach would require us to regard a brain as having its own way of behaving, even in isolation, and to ask how this behavior is modified by a stream of stimuli from its environment. A very simple model of this sort is proposed.

2. Definition of a network

From the point of view of someone making a mathematical model, a neural network is an assembly of objects (cells) with the following properties:

(i) at a given time, a cell can be active (firing) or inactive;

(ii) cells are connected by paths; a pair of cells A, B may be unconnected, or connected by any number of paths; these paths are directional;

(iii) if there is a path $\overrightarrow{AB}$, the firing of A at some time may contribute to the firing of B at a later time; A may be described as an input cell for B;

(iv) a cell fires either as the result of an external stimulus or because of the firing of its input cells.

Various details about these properties must be specified in the model. The inactive state referred to in (i) is usually classified into two states (a) used, for some period after the cell has fired, during which it cannot fire again (a recovery time or refractory period), (b) sensitive, when the cell can be reactivated. In (iii) the time for a signal to go from A to B (the synaptic delay) must

be given; if this time is δ, then it is usually assumed that the firing of A at time t can only affect the state of B at time $t + \delta$. In discrete time models δ is taken as the unit of time and the refractory period is taken either as less than δ or equal to some multiple of it. The number of paths, or a probability distribution for the number must be given under (ii). For (iv), we need to know the minimum number θ of input cells which must fire together if a cell is to become active; $\theta(\geqq 1)$ is the threshold of the receiving cell and may also be described by a probability distribution.

A cell whose firing may stimulate another cell provides an excitatory input. It is also possible for a cell to provide an inhibitory input which discourages the receiving cell from firing. If this is included in the model then the receiving cell will fire only if it has more excitatory inputs than inhibitory ones, and the exact number more which is required must be given.

The properties summarized above are based on neurological data; descriptions of the experimental work can be found in Sherrington [54], Adrian [1], [2], for example.

It will be seen that models of neural networks have much in common with models for the spread of infection and with birth and death processes. In fact, some of the results that have been obtained for networks are well known results in epidemiology and genetics.

3. An early model

Rashevsky ([47], chapter 22; the first edition is cited deliberately) aimed to produce "a systematic mathematical biology, similar in its structure and aims to mathematical physics" so that it is not surprising that he works mainly with integral and differential equations. He assumes that a firing cell builds up excitatory and inhibitory factors (e and i) in paths connecting it to an adjacent cell at rates given by

$$\begin{aligned} \frac{de}{dt} &= AE - ae, \\ \frac{di}{dt} &= BE - bi, \end{aligned} \tag{3.1}$$

where A, B, a, and b are constant and E is the effect of an external stimulus; if $e > i$ at any time then $(e - i)$ acts as the E for the next cell. E is assumed to be of the form

$$E \propto \frac{1}{\tau}\left[1 - e^{-\alpha\tau(S-\theta)}\right], \qquad E = 0 \quad \text{if} \quad S < \theta, \tag{3.2}$$

where τ is the refractory period, S is the external stimulus, θ is the threshold, and α is constant. This form is chosen so that $E \propto 1/\tau$ for S large. The application of a stimulus to a neuron in fact produces a train of pulses of a frequency which increases with the strength of the stimulus, and this frequency must have an upper limit $1/\tau$. However E is treated as a continuous variable. Rashevsky

discusses the cases of excitatory and inhibitory paths operating separately and together and shows that the synaptic delay will depend on the input stimulus as well as on the characteristics of the cells.

Rashevsky examines, for a chain of cells, the way in which a steady input stimulus S is transformed at different points of the chain. He also obtains (but does not solve) equations for an input varying with time and says that one could determine a sequence of stimuli giving maximal central stimulation. In chapter 24 he considers a closed circuit of two cells (without inhibition) and finds that there is a stable solution that "can be regarded as a source of spontaneous nerve activity in the absence of external stimulation."

The main difference between this and later work is that continuous functions are introduced more or less intuitively and *ad hoc* and the discrete nature of the response of a cell is virtually disregarded. Nowadays it is more usual to discuss a discrete model in the first place, even if a continuous approximation is all that can be solved readily.

4. A logical model

McCulloch and Pitts [38] use the fact that the cell must be in one of two states to describe the network by means of the propositional calculus. This is a discrete time model, time being measured by the number of synaptic delays, and assumes inhibitory effects. They show that a complete specification of the net, that is, cells and their connections, excitatory or inhibitory, and their thresholds, enables one to compute the state of the net at time $t + 1$ given the state and the external stimuli at time t; and they discuss the relation of such a net to a Turing machine. This seems to be the earliest paper in which a neural network is compared with a digital computer.

The model is, of course, completely adequate provided that (a) the specification of the net does not change with time and (b) the input from external stimuli can be described exactly. A real brain, supported by a real body, might be affected by lack of oxygen or food, which would perhaps change the threshold levels of cells; the input from the environment to, say, a real visual system can usually only be described in probabilistic terms. It is the statistical nature of the input from the environment that makes a statistical model more desirable, though such a model would have the added advantage that it could be described much more economically.

5. Statistical models (1) randomly connected nets

One of the earliest of these was that of Shimbel and Rapoport [56]. They use a discrete time model, without inhibitory effects, and assume that cells are connected at random and that there is a given distribution of thresholds. A similar model was considered by Ashby *et al.* [5], using a fixed threshold and a fixed number of inputs to each cell. In neither paper is the size of the net speci-

fied, and results are worked out in terms of the proportion of cells firing at a given time. If we include in the argument the fact that the number of cells is N, fixed (which can mean either that we consider the whole net at successive times or successive pieces of the net, each of the same size), then, if j cells fire at time t,

$$\phi_j = P\{\text{cell in set next available fires}\} = \sum_{i=\theta}^{n} \binom{n}{i} \left(\frac{j}{N}\right)^i \left(1 - \frac{j}{N}\right)^{n-i}, \tag{5.1}$$

where n is the number of input cells, θ is the threshold number (both fixed);

$$\phi_j = \begin{matrix} 0 & \text{if} & j < \theta, \\ 1 & \text{if} & j = N. \end{matrix} \tag{5.2}$$

The process is then a Markov chain with

$$p_{jk} = \binom{N}{k} \phi_j^k (1 - \phi_j)^{N-k}. \tag{5.3}$$

If $n = \theta = 1$ the model is the same as Feller's [17] for the transmission of genes by random mating in a population of fixed size. For this case (and clearly also for the case of other values of n and θ), 0 and N are absorbing states; Ashby *et al.* say that, whatever the input, the "brain" would ultimately be in a state of coma or epilepsy, and regard this a a paradox.

Shimbel and Rapoport give a more sophisticated treatment of this problem (they consider how many paths to a given cell come from the same input cell) but essentially reach the same result with the special cases they consider. In fact, in models of this sort it is clear that, however complicated the details are, ultimately one ends up with a transition matrix p_{jk} for the probability of changing from j active cells to k active cells, with $p_{NN} = p_{00} = 1$.

Beurle ([8], appendix a) has a model for which 0 is an absorbing state but N is not; this is because he considers a longer refractory period so that following an active state there is a used state. Consequently, if at any time all cells are active, at the next instant all will be used and the excitation will die out. Beurle's formulation is not easy to follow, and his conclusion that, if the number firing at any time is large, then the number firing at the next instant will be larger seems to be wrong (since the number of sensitive cells would be small). We may formulate his model in another way, using continuous time. Let a, u, s be the number active, used, sensitive cells at time t, let λ, ρ, μ, be the rates at which an active cell becomes used, a used cell becomes sensitive, and a sensitive cell becomes active; μ will depend on a, $\mu = \kappa a$ say. Then, if

$$\Pi(x, y, z; t) = \sum_{a+u+s=N} p(a, u, s; t) x^a y^u z^s, \tag{5.4}$$

Π satisfies the equation

$$\frac{\partial \Pi}{\partial t} = \lambda(y - x)\frac{\partial \Pi}{\partial x} + \rho(z - y)\frac{\partial \Pi}{\partial y} + \kappa x(x - z)\frac{\partial^2 \Pi}{\partial x\, \partial z}. \tag{5.5}$$

For the mean values, m_a, m_u, m_s, we have

$$\begin{aligned} \frac{dm_a}{dt} &= -\lambda m_a + \kappa E(as), \\ \frac{dm_s}{dt} &= \rho m_u - \kappa E(as), \end{aligned} \tag{5.6}$$

and $m_a + m_u + m_s = N$. The corresponding deterministic equations, using a and s for the proportion active and sensitive, are

$$\begin{aligned} \frac{da}{dt} &= -\lambda a + kas, \\ \frac{ds}{dt} &= \rho(1 - a - s) - kas. \end{aligned} \tag{5.7}$$

Here, k replaces κN but is not necessarily equal to it since the chance that an active cell stimulates a sensitive one might well decrease with the number of cells. These equations could represent the course of an endemic infection in which a was the proportion infected, s the proportion susceptible, $1 - a - s$ the proportion recovered and temporarily immune. We should still expect the infection to die out ultimately but can nevertheless investigate whether a temporary state of stable equilibrium is possible. (A different model is considered in Bailey [7], p. 136.)

The equilibrium solution is $a_0 = \rho(\rho + \lambda)^{-1}(1 - \lambda/k)$, $s_0 = \lambda/k$, and we require $\lambda < k$. Putting $a = a_0(1 + v)$, $s = s_0(1 + u)$ and neglecting uv,

$$\frac{d^2u}{dt^2} + \frac{\rho(\rho + k)}{(\rho + \lambda)} \frac{du}{dt} + \rho(k - \lambda) = 0, \tag{5.8}$$

so that the equilibrium position will be stable if

$$\rho(\rho + k)^2 < 4(k - \lambda)(\rho + \lambda)^2 \tag{5.9}$$

and this will certainly be true if

$$\rho < 4(k - \lambda)\lambda^2/k^2; \tag{5.10}$$

for instance, for the common cold we might have $\rho = \lambda = 3k/5$ and $a_0 = 1/5$, $s_0 = 3/5$. The constant k might represent the number of encounters between people, or the number of connecting paths between cells, and so k could be almost independent of N.

It appears in general that, in a randomly connected network with only excitatory inputs, the effect of an external stimulus will ultimately die out if there is a long recovery time for cells, and will either die out or flare up if there is a recovery time less than the synaptic delay. If inhibitory inputs are included in the model, it would seem plausible that this would simply increase the probability that a process would die out.

No one seems to have asked how long the effect would take to die out (or flare up). If successive times relate to different sections of the net and there were a finite number of sections, a reasonable approximation to the stimulus input

might be transmitted. The work of Darroch and Senata [12] and Senata [53] on quasistationary distiributions in genetcs would seem to be applicable to this problem.

6. Statistical models (2) nonrandom connections

In order to avoid this trouble—that is, that in a randomly connected net all cells are ultimately in the same state—Griffith [20] suggested various forms of deterministic nets. These models are statistical because he allows for the failure of components (cells or paths) with given probability. This problem was originally studied by von Neumann, who discussed the use of error correcting codes to compensate for faulty units. Winograd and Cohen [72] have continued this work and give useful summaries and references.

7. Connection of network theory with models in genetics and epidemiology

It was mentioned earlier that with random connectivity, each cell having only one input path, and each cell having a threshold of unity, the transition matrix for the number of cells firing at successive times was the same as that for the number of one allele of a gene with two alleles in a population with random mating. The number of cells and the size of the population must both remain fixed. (The case where the number of input paths is greater and the threshold higher would correspond in genetics to multiple parentage with a rule about dominance and has, perhaps naturally, not been considered by geneticists.) Spontaneous firing or spasmodic failure of a cell would correspond to the two sorts of mutation possible. If there is mutation in one direction only, the population ultimately becomes homozygous (absorption at 0 or N); with mutation in both directions it is reasonable to look for a stationary distribution.

This problem has been discussed by, for example, Moran [40] and Feller [17] in terms of diffusion processes; the steady state solution depends, as one would expect, only on the mutation rates.

Alternatively, a firing cell corresponds to an infected person, a sensitive cell to a susceptible person, in an epidemic model. The field theories of Beurle and Griffith resemble models for the spread of infection when the effect of geographical location is taken into account (and similar models in genetics). In particular, Beurle's equations leading to plane waves are essentially the equations for a linear epidemic. (Beurle omits the effect due to the recovery of used cells and apart from this his second equation is wrong.) For a useful summary of models of the spread of infection see Kendall [29]. Griffith [21] looks for a partial differential equation to relate the amount of activity at a point f with the flow of excitation towards the point ψ and by arguments rather like those of Rashevsky obtains a second order equation, the simplest form of which has a stable solution ψ = constant; he does not discuss boundary conditions. Presumably, if there is

an initial disturbance and then no further stimulation, the constant will be zero on the boundary and therefore throughout the network.

8. Coral colonies

Horridge [27] investigates the behavior of colonies of coral polyps in which each polyp behaves like a cell, with all or none firing properties and the ability to transmit stimulation to neighboring cells. If a single polyp is stimulated electrically it retracts and so do others in its neighborhood. Each time the stimulation is repeated more polyps retract and the effect spreads further from the origin. The rate of spreading depends on the species. To explain this Horridge proposes a model which includes interneural facilitation. He assumes that neighboring cells are linked by paths which are two way, although excitation only moves away from a stimulated cell, not back to it again. A path can be conducting or nonconducting. If a cell fires, all cells connected to it by conducting paths also fire and all nonconducting paths from it become conductive by the time of the next firing of the cell.

Horridge produces results for the spread of excitation after successive stimuli by Monte Carlo methods (manipulating pegs on a board with holes in it). The amount of spread depends on the proportion of pathways initially conducting and on the number of neighbors to which a cell is connected (he considers the cases of three connections and six connections). This model is a branching process in which branches may coalesce, or a percolation process as treated by Broadbent and Hammersley [9]. Hammersley in later papers [22], [23] shows that there is a critical proportion of initially conducting paths below which excitation will not spread across the whole network.

An approximate evaluation of the critical proportion may be obtained by treating the flow of excitation as a spread of infection. For the sort of networks considered by Horridge we may define successive rings about the point of stimulation as follows: if all connecting paths are conducting, then the first ring consists of all cells reached by a single step from the point of stimulation zero, where taking a step means traversing one path; the second ring consists of those cells not previously reached which can be reached in two steps; and so on. The rings are approximately concentric circles and the nth ring has αn cells in it, where α is the number of paths from each cell. (Horridge considers $\alpha = 3, 6$.) Take as the unit of length the distance between rings and as unit of time the time to traverse a path. Regard a cell as susceptible if it has not yet fired and as inert if it has just fired. Then if $x(r, \theta; t)$, $y(r, \theta; t)$ are the densities of susceptible and active cells, respectively, at the point (r, θ) at time t, we have approximately

$$y(r, \theta; t + 1) = (\alpha - 1)px(r, \theta; t)\bar{y}(r, \theta; t), \tag{8.1}$$

where $\bar{y}$ is the local mean of y and p is the probability that a path is connecting;

an active cell has (at most) $\alpha - 1$ paths along which it may send excitation. We thus have approximate deterministic equations

$$\begin{aligned} \frac{\partial y}{\partial t} &= (\alpha - 1)px\bar{y} - y, \\ \frac{\partial x}{\partial t} &= -(\alpha - 1)px\bar{y}, \end{aligned} \tag{8.2}$$

corresponding to Kendall's [29] equations for a linear epidemic. If we assume from symmetry that y is independent of θ then the same treatment as Kendall's may be applied to these equations to get the result that a circular wave traveling outwards is possible only if $p > (\alpha - 1)^{-1}$. For a tripolar net this would require $p > 1/2$, for a hexipolar net it would require $p > 1/5$; Horridge, in fact, finds through conduction occurring at these values of p.

For small p, Horridge's data for the total number of cells fired appears to be approximated quite well by taking for each of the α paths from the point of stimulation a separate one dimensional process, in which the probability of the first step is p and of each further step is $(\alpha - 1)p$. For one path, the probability generating function for the number of steps from the origin is

$$1 - p + zp\frac{1 - (\alpha - 1)p}{1 - (\alpha - 1)pz} \tag{8.3}$$

and the mean number of cells fired over all α paths, and including the origin, is $(1 + p)[1 - (\alpha - 1)p]^{-1}$. The predicted mean number and variance and the observed mean number and variance from Horridge's data are shown in table I.

TABLE I

PREDICTED MEAN NUMBER AND VARIANCE AND OBSERVED MEAN NUMBER AND VARIANCE FROM HORRIDGE'S DATA

		Predicted		Observed	
α	p	Mean	Variance	Mean	Variance
3	0.1	1.375	0.52	1.4	0.4
3	0.2	2.0	2.0	1.9	1.8
3	0.3	3.25	7.3	3.2	9.0
6	0.1	2.2	3.4	2.4	3.1
6	0.15	4.6	23.2	3.6	12.5

This method of calculating the mean number of cells fired ignores the chance of counting the same cell twice or more and also the chance of one cell affecting two or more cells; it appears that these two effects more or less cancel out.

9. Clutches of eggs

Eggs of game birds incubated in a clutch tend to hatch all at the same time, even eggs added to the clutch twenty four hours late will hatch at about the

same time as the rest of the clutch. An isolated egg can be hurried up by vibration or sound, so that it is probable that one egg influences another by means of the clicks made by the chick in the egg, or by the movements accompanying the clicks. For an account of the experimental work see Vince [69].

We may regard an egg as a cell and a group of eggs in contact as a neural network. The eggs may be laid out in various patterns; for the moment it is assumed that every egg touches every other egg (which may be arranged by, for example, joining two eggs not in physical contact by a wire). This corresponds to a network in which every pair of cells has two connecting paths, one in each direction.

Each egg can be either active, that is, clicking, or inactive. If we work with discrete time, using as the unit interval the minimum time between clicks, and write 1 for the active state and 0 for the inactive state, we may write down transition probabilities for a single egg as follows:

$p_{01} = 1$ if some other egg is active in the preceding interval,
$p_{01} = p$ otherwise,
$p_{11} = p$ always.

These are based on the assumptions that the egg will click spontaneously with probability p and that, if it is inactive at any instant, it will be able to receive a signal from another egg, to which it will respond at the next instant. For a set of M eggs, all touching each other, we can evaluate $p_{jk} = P\{j \text{ active followed by } k \text{ active}\}$ and get a Markov chain which is finite, accessible and aperiodic. The generating function for time n

$$G_n(z) = \sum_0^M p_k(n) z^k \tag{9.1}$$

satisfies the equation

$$G_{n+1}(z) = z^M G_n(p + qz^{-1}) + G_n(0)[(pz + q)^M - z^M]. \tag{9.2}$$

We can obtain the steady state solution $G_\infty(z)$ as follows. If M is large, $p_0(n)$ is small. Further, if we neglect $p_0(n)$ we shall have M eggs acting independently, each with transition matrix determined by $p_{01} = 1$, $p_{11} = p$, and with a steady state generating function proportional to $(q + z)$. Therefore $(q + z)^M$ should be an approximation to $G_\infty(z)$. For M independent eggs the state $k = 0$ is automatically followed by the state $k = M$. If we adjust the solution for this by taking $G_\infty(z) = (q + z)^M - q^M z^M$ we find that it satisfies (9.2). Using the condition $G_\infty(1) = 1$, we find that the steady state solution is

$$[(q + z)^M - q^M z^M][(q + 1)^M - q^M]^{-1}. \tag{9.3}$$

Thus if M is large and p is nonzero we can in fact treat the M eggs as acting independently to a good degree of approximation; the case of independence corresponds to there being an extra, always active, egg which acts as a pacer to the rest. For this case, the mean number active at time $2n$ if r are active at time 0 is

$$\frac{M}{1+q} + \left(r - \frac{M}{1+q}\right) q^{2n}, \tag{9.4}$$

and this gives some idea of the rate of approach to the steady state.

If the ith egg has probability p_i of clicking spontaneously, the corresponding steady state solution is proportional to

$$\prod_{i=1}^{M} (q_i + z_i) - \prod_{i=1}^{M} q_i z_i \tag{9.5}$$

and the probability that the ith egg is active is ultimately $(1 - \alpha q_i)(1 + q_i)^{-1} = P_i$ say, where

$$\alpha = \prod_{i=1}^{M} q_i \left[\prod_{j=1}^{M} (q_j + 1) - \prod_{j=1}^{M} q_j \right]^{-1}. \tag{9.6}$$

If $\delta\mu$ and σ^2 are the mean and variance of the p_i, the variance of P_i is approximately $\sigma^2(1 + \alpha)^2(2 - \mu)^{-4}$. Since α is small the difference in rate of clicking among the eggs will be diminished; in fact, all will be speeded up but the slower ones will have a greater change in rate. The time of hatching appears to be related to some extent to the amount of clicking that has occurred, so that an interaction of this sort between eggs would account for some reduction in the range of hatching times.

This is a very simple statistical model describing the effect of environment on a cell capable of spontaneous activity. In the case where the number of other cells is large the environment is approximately constant.

10. Brains and computers

In designing a computer we require that it should handle determinate input signals accurately, transmitting or transforming them according to instructions, that it should use the minimal number of cells and connections consistent with accuracy, and that it should be at rest when we do not use it. Brains have to handle inputs subject to statistical variation, they respond most readily to changes in the environment and often habituate to a statistically steady environment, and, as electroencephalographic records show, are never at rest; most brains contain a large number of cells and probably use them wastefully. If we wish to understand how a brain works it is better to look for a model that has some of the properties of a brain. In the next section a very simple model of this sort is described. It is based on some of the facts known about vision. Psychophysical experiments undoubtedly show that a statistical model is appropriate in this case (see, for example, Gregory [19]).

11. A simple model for a brain

Suppose that there are two sorts of cells, C cells (thought of as cortical) with a rapid recovery time and R cells (thought of as receptors) with a longer recovery

time. C cells behave like eggs in that they can excite each other, but, instead of having spontaneous activity, they are otherwise excited by the input from R cells. Suppose that each C cell is linked to an R cell (the path being from R to C) and that all C cells are connected together both ways. Take discrete time intervals, corresponding to synaptic delay times, and suppose that a C cell can be active at two successive times but that an R cell must be inactive for one time interval after it has been active; after this it becomes sensitive again. If we regard a steady stimulus as something which causes an R cell to fire with probability p if it is sensitive then the transition matrix for the R cell is determined by $p_{01} = p$, $p_{11} = 0$. If the number of C cells is large, so that we can approximately treat them as independent, then the transition from the state of an $R - C$ pair at time n to the state at time $(n + 1)$ can be written

$$(11.1) \qquad (p_{00}p_{01}p_{10}p_{11})_n \begin{pmatrix} 0 & q & 0 & p \\ q & 0 & p & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 1 & 0 & 0 \end{pmatrix} = (p_{00}p_{01}p_{10}p_{11})_{n+1},$$

where p_{ij} means R in state i, C in state j. In assuming independence of cell pairs we are neglecting the case when all C cells are inactive; this can only happen at time $(n + 1)$ if at time n either (i) all cell pairs (M of them, say) are in state 00 or (ii) all cell pairs are in state 01. The probability of these two cases together is less than P {all R cells inactive at n} $\to 1/(1 + p)^M$ as $n \to \infty$.

The steady state solution for an $R - C$ pair is proportional to

$$(11.2) \qquad (q + 1 + p + pq)(1 + p)^{-1}(1 + q)^{-1},$$

that is, $P\{R \text{ active}\} = p(1 + p)^{-1}$, as we should expect, and $P\{C \text{ active}\} = (1 + pq)(2 + pq)^{-1}$. Note that the latter is symmetric in p and q and varies very little (from 1/2 to 5/9). If there are M cells of type C, the mean number active is ultimately $M(1 + pq)(2 + pq)^{-1}$, with variance $M(1 + pq)(2 + pq)^{-2}$; the latter is very close to $M/4$ for all values of p. The rate of progress to the steady state depends mainly on $\theta^n(p - q)^{-1}$, where $\theta = \max(p, q)$, or on $n(1/2)^n$ if $p = 1/2$. For vision in normal light p is likely to be less than 1/2 and so this will be assumed. If, when input ceases, there are kC cells active and p is zero thereafter, the number of active C cells would oscillate between k and $N - k$, that is, a residual effect from the previous signal would persist. This seems unrealistic, so it is supposed that p has some small value, δ say, which may correspond either to some slight external stimulation or to spontaneous firing in either the R cells, the C cells, or both; the actual source is mathematically almost irrelevant. Then after a long period of rest about half of the C cells would fire at each instant.

Now suppose that at time t the system is in a steady state under input p_0 and that at $t + 1$ the input is changed to $p_0 + \Delta$. For a given C cell,

$$P\{\text{active at } t\} = (1 + p_0q_0)(2 + p_0q_0)^{-1},$$
$$P\{\text{active at } t + 2\} = (1 + p_0q_0 + q_0\Delta)(2 + p_0q_0)^{-1},$$
$$P\{\text{active at } t + 3\} = (1 + p_0q_0 - p_0\Delta - \Delta^2)(2 + p_0q_0)^{-1},$$

so that the effect of the change is to set up an oscillation about the previous steady-state; we may get a reverse effect by switching back to p_0 after the system has more or less reached the steady state for $p_0 + \Delta$. Thus in a state of steady stimulation the system settles down (and the expected number of C cells active at any instant would be close to $M/2\delta$, whereas a change in state sets up a fluctuation which gradually dies away. (This corresponds roughly to the observed change in the frequency of spike potentials in an optic nerve.) Such an oscillation could be detected by a detector cell, D say, which was connected to all the C cells and had a threshold $M/2 + \alpha\sqrt{M}/2$, for a suitable constant α. There could indeed be a series of such cells with different α values. An increase in input Δ would usually be detected if

$$\frac{M(1 + p_0q_0 + q_0\Delta)}{2 + p_0q_0} > \frac{M}{2} + \frac{\alpha M^{1/2}}{2}, \tag{11.3}$$

that is, approximately if

$$\Delta > \alpha M^{-1/2} + \frac{1}{2}p_0(3\alpha M^{-1/2} - 1). \tag{11.4}$$

For example if $\alpha = 2$, and $M < 36$, this would give that the just noticeable difference is linearly related to the background intensity (an observed result for visual thresholds).

If an $R - C$ pair is in a steady state under spontaneous firing (or weak external stimulation) so that approximately

$$(p_{00}p_{01}p_{10}p_{11}) = \frac{1}{2 + \delta}(1 - \delta, 1, \delta, \delta) \tag{11.5}$$

and an input p is switched in for one time interval only, so that in the next interval the input is δ again, the probabilities for the four states will change, over the two intervals, to

$$\frac{1}{2 + \delta}[q(1 - \delta), (1 + p + \delta), q\delta, q\delta] \tag{11.6}$$

and $P\{C \text{ active}\}$ changes from about $1/2$ to about $(1 + p)/2$. In general if the initial state is

$$\frac{1}{1 + \epsilon + \eta}(x, 1 - x, \epsilon, \eta) \tag{11.7}$$

where ϵ, η are $O(\delta)$, then after the same two inputs $P\{C \text{ active}\}$ changes from about $1 - x$ to about $1 - xq$; with repeated alternation of inputs of this kind we get $P\{C \text{ active}\}$ increasing to 1. Thus a flicker at the right rate can cause increasing oscillation of the number active in a way that steady stimulation cannot, and this may be related to the way in which a flickering light can induce an epileptic seizure. The effect described above occurs because the receptors just have time to recover from one stimulus before the next is applied; the details of the model are irrelevant.

This model has multiple, nonrandom connections (so that a moderate amount

of component failure would not matter), differential recovery rates, and no absorbing states; an input signal would ultimately die out, but this seems desirable.

12. Discussion and summary

A model should provide a convenient way of thinking about a real problem. There are two problems for which a network model is required:

(i) to discuss the way in which excitation flows through nerve tissue;

(ii) to discuss the behavior of a brain as a whole.

The complexity of connection of actual nerve cells, and the fact that input from the environment is never determinate, imply a statistical treatment and a number of the models reviewed here are based on randomly connected cells; such models are essentially concerned with problem (i). One may deduce from them that, if thresholds are low and recovery time short, excitation will either flare up or die out; if thresholds are high or recovery time is long, or if inhibition is introduced into the model, then excitation will die out. It is clear that such a decay is necessary, for, in a randomly connected net, the fact that a signal has reached its target will not ensure that it is removed from the system. What needs investigation is the question of how long a signal persists in a recognizable form. Moreover, if a detailed representation of the flow of excitation is required, the simple assumptions of all or none firing and random connectivity may have to be replaced by more complex ones.

The only attempts to deal with problem (ii) directly, that I know of, are concerned with learning. Learned behavior is, however, a modification of existing behavior and we should begin by finding a way of representing the simplest behavior possible. For problem (ii) we need some more organized form of connectivity, but in other ways the model can be much cruder because we are concerned with macroscopic effects. I have proposed here a very simple model which might represent the response of a visual system to light.

I should like to express my most grateful thanks to Newnham College, Cambridge, and Mr. I. Behrens, for the award of a grant which relieved me of some teaching duties; to Dr. F. J. MacWilliams, of the Bell Telephone Laboratories, with whom I worked on this and other problems, and to the Department of Scientific and Industrial Research, who awarded her a Senior Visiting Fellowship so that she could come to Cambridge; and to Mr. E. Thomas, who gave me a great deal of help in collecting the references.

REFERENCES

[1] E. D. ADRIAN, "The control of nerve-cell activity, *Physiological Mechanisms in Animal Behaviour*, Cambridge, Cambridge University Press, 1950.

[2] ———, *Physical Mechanism of Nervous Action*, Philadelphia, University of Pennsylvania Press, 1932.

[3] J. T. ALLANSON, "Some properties of randomly connected neural networks," *Third London Symposium on Information Theory*, London, Butterworth, 1956, pp. 303–313.
[4] W. R. ASHBY, "Design for a brain," *Electronic Engng.*, Vol. 20 (1948), pp. 379–383.
[5] W. R. ASHBY, H. VON FOERSTER, and C. C. WALKER, "Instability of pulse activity in a net with threshold," *Nature*, Vol. 196 (1962), pp. 561–562.
[6] H. B. BARLOW, "The information capacity of nervous transmission," *Kybernetik*, Vol. 2 (1963), p. 1.
[7] N. T. J. BAILEY, *The Mathematical Theory of Epidemics*, London, Griffin, 1957.
[8] R. L. BEURLE, "Properties of a mass of cells capable of regenerating pulses," *Philos. Trans. Roy. Soc. London Ser. B*, Vol. 240 (1956), pp. 55–94.
[9] S. R. BROADBENT and J. M. HAMMERSLEY, "Percolation processes, I. Crystals and mazes," *Proc. Camb. Philos. Soc.*, Vol. 53 (1957), pp. 629–641.
[10] B. D. BURNS, "The mechanism of after-bursts in cerebral cortex," *J. Physiol., Lond.*, Vol. 127 (1955), pp. 168–188.
[11] J. U. CASBY, R. SIMINOFF, and T. R. HOUSEKNECHT, "An analogue cross-correlator to study naturally induced activity in intact nerve trunks," *J. Neurophysiol.*, Vol. 26 (1963), pp. 432–448.
[12] J. N. DARROCH and E. SENETA, "On quasi-stationary distributions in absorbing discrete-time finite Markov chains," *J. of Appl. Probability*, Vol. 2 (1965), pp. 88–100.
[13] J. C. ECCLES and A. K. MCINTYRE, "Plasticity of mammalian monosynaptic reflexes," *Nature*, Vol. 167 (1951), pp. 466–468.
[14] J. C. ECCLES, *The Neurophysiological Basis of Mind*, Oxford, Clarendon Press, 1953.
[15] ———, *The Physiology of Synapses*, Berlin, Springer-Verlag, 1964.
[16] B. G. FARLEY, L. S. FRISHKOPF, W. A. CLARK, JR., J. T. GILMORE, JR., "Computer techniques for the study of patterns in EEG," *IRE Trans. Biomed. Electronics*, Vol. BME-9 (1962), pp. 4–12.
[17] W. FELLER, "Diffusion processes in genetics," *Proceedings of the Second Berkeley Symposium on Mathematical Statistics and Probability*, Berkeley and Los Angeles, University of California Press, 1951, pp. 227–246.
[18] G. L. GERSTEIN and B. MANDELBROT, "Random walk models for the spike activity of a single neuron," *Biophys. J.*, Vol. 4 (1964), pp. 41–68.
[19] R. GREGORY, "An experimental treatment of vision as an information source and noisy channel," *Third London Symposium on Information Theory*, London, Butterworth, 1956, pp. 287–299.
[20] J. S. GRIFFITH, "On the stability of brain-like structures," *Biophys. J.*, Vol. 3 (1963), pp. 299–308.
[21] ———, "A field theory of neural nets: II" *Bull. Math. Biophys.*, Vol. 27 (1965), pp. 187–195.
[22] J. M. HAMMERSLEY, "Percolation processes," *Ann. Math. Statist.*, Vol. 28 (1957), pp. 790–795.
[23] ———, "Percolation processes, II," *Camb. Phil. Soc.*, Vol. 53 (1957), pp. 642–645.
[24] L. D. HARMON, *Natural and Artificial Synapses, in Self-Organising Systems*, Washington, Spartan, 1962, pp. 177–202.
[25] F. F. HILTZ, "Analog computer simulation of a neural element," *IRE Trans. Biomed. Electronics*, Vol. BME-9 (1962), pp. 12–20.
[26] A. L. HODGKIN and A. F. HUXLEY, "A quantitative description of membrane current and its application to conduction and excitation in nerve," *J. Physiol. Lond.*, Vol. 177 (1952), pp. 500–544.
[27] G. A. HORRIDGE, "The co-ordination of the protective retraction of coral polyps," *Phil. Trans. Roy. Soc. London Ser. B*, Vol. 240 (1956), pp. 495–528.
[28] A. S. HOUSEHOLDER and A. D. LANDAHL, *Mathematical Biophysics of the Central Nervous System*, Bloomington, Principia Press, 1945.
[29] D. G. KENDALL, "Mathematical models of the spread of infection," *Mathematics and*

Computer Science in Biology and Medicine, Medical Research Council, Great Britain, 1965, pp. 213–225.

[30] H. D. LANDAHL and R. RUNGE, "An outline of a matrix algebra for neural nets," *Bull. Math. Biophys.*, Vol. 8 (1946), pp. 75–81.

[31] H. D. LANDAHL, "A matrix calculus for neural nets, II," *Bull. Math. Biophys.*, Vol. 9 (1947), pp. 99–108.

[32] K. LASHLEY, "Studies of cerebral function in learning VII," *J. Comp. Neurol.*, Vol. 41 (1926), pp. 1–58.

[33] R. LORENTE DE NÓ, "A study of nerve physiology I and II," *Studies from the Rockefeller Institute for Medical Research*, Vols. 131 and 132, New York, 1947.

[34] D. M. MACKAY and W. S. MCCULLOCH, "The limiting information capacity of a neuronal link," *Bull. Math. Biophys.*, Vol. 14 (1952), pp. 127–135.

[35] D. M. MACKAY, "Modelling of large-scale nervous activity," *Models and Analogues in Biology*, Symposia of the Society for Experimental Biology, No. 14, Cambridge, Cambridge University Press, 1960, pp. 192–198.

[36] R. MAYNE, "Some engineering aspects of the mechanism of body control," *Electl. Engng.*, Vol. 70 (1951), pp. 207–212.

[37] D. M. MCCALLUM and J. B. SMITH, "Mechanical reasoning, logical computers and their design," *Electronic Engng.*, Vol. 23 (1951), pp. 126–133.

[38] W. S. MCCULLOCH and W. PITTS, "A logical calculus of the ideas immanent in nervous activity," *Bull. Math. Biophys.*, Vol. 5 (1943), pp. 115–132.

[39] H. MCLENNAN, *Synaptic Transmission*, Philadelphia, Saunders, 1963.

[40] P. A. P. MORAN, *The Statistical Processes of Evolutionary Theory*, Oxford, Clarendon Press, 1962.

[41] J. NAGAMO, S. ARIMOTO, and S. YOSHIGAWA, "An active pulse transmission line simulating a nerve axon," *Proc. I.R.E.*, Vol. 50 (1962), pp. 2061–2070.

[42] A. RAPOPORT, "Contributions to the probabilistic theory of neural nets: I. Randomisation of refractory periods and of stimulus intensity," *Bull. Math. Biophys.*, Vol. 12 (1950), pp. 109–120.

[43] ———, "Contributions to the probabilistic theory of neural nets. II. Facilitation and threshold phenomena," *Bull. Math. Biophys.*, Vol. 12 (1950), pp. 187–197.

[44] ———, "Nets with distance bias," *Bull. Math. Biophys.*, Vol. 13 (1951), pp. 85–91.

[45] ———, "Ignition phenomena in random nets," *Bull. Math. Biophys.*, Vol. 14 (1952), pp. 35–44.

[46] ———, "Response time and threshold of a random net," *Bull. Math. Biophys.*, Vol. 14 (1952), pp. 351–363.

[47] A. RAPOPORT and R. SOLOMONOFF, "Connectivity of random nets," *Bull. Math. Biophys.*, Vol. 13 (1951), pp. 107–117.

[48] N. RASHEVSKY, *Mathematical Biophysics*, Chicago, University of Chicago Press, 1938.

[49] ———, "Some bio-sociological aspects of the mathematical theory of communication," *Bull. Math. Biophys.*, Vol. 12 (1950), pp. 359–378.

[50] J. B. ROBERTS, "Mathematical biophysics of some neural nets," *Bull. Math. Biophys.*, Vol. 10 (1948), pp. 1–10.

[51] ———, "The group structure of some neural nets," *Bull. Math. Biophys.*, Vol. 11 (1949), pp. 51–58.

[52] R. W. RODIECK, N. Y.-S. KIANG, and G. L. GERSTEIN, "Some quantitative methods for the study of spontaneous activity of single neurons," *Biophys. J.*, Vol. 2 (1962), pp. 351–368.

[53] E. SENETA, "Quasi-stationary distributions and time-reversion in genetics," *J. Roy. Statist. Soc. Ser. B*, Vol. 28 (1966), pp. 253–277.

[54] C. S. SHERRINGTON, *Integrative Action of the Nervous System*, New Haven and London, Yale University Press, 1906.

[55] ———, "Inhibition as a co-ordinative factor," Nobel Lecture 1932, Stockholm, P. A. Norstedt, 1934.
[56] A. SHIMBEL and A. RAPOPORT, "A statistical approach to the theory of the central nervous system," *Bull. Math. Biophys.*, Vol. 10 (1948), pp. 41–55.
[57] A. SHIMBEL, "An analysis of theoretical systems of differentiating nervous tissue," *Bull. Math. Biophys.*, Vol. 10 (1948), pp. 131–143.
[58] ———, "Contributions to the mathematical biophysics of the central nervous system with specific reference to learning," *Bull. Math. Biophys.*, Vol. 12 (1950), pp. 241–275.
[59] D. A. SHOLL, "Dendritic organisation in the neurons of the visual and motor cortices of the cat," *J. Anat.*, Vol. 87 (1953), pp. 387–406.
[60] R. SIMINOFF and T. R. HOUSEKNECHT, "An analog cross-correlator for the analysis of physiological transducers," *Med. Electron. Biol. Engng.*, Vol. 2 (1964), pp. 57–69.
[61] D. R. SMITH and C. H. DAVIDSON, "Maintained activity in neural nets," *J. Assoc. Comput. Mach.*, Vol. 9 (1962), pp. 268–279.
[62] G. K. SMITH and D. R. SMITH, "Spike activity in the cerebral cortex," *Nature*, Vol. 202 (1964), pp. 253–255.
[63] W. K. TAYLOR, "Computers and the nervous system," *Models and Analogues in Biology*, Cambridge, Cambridge University Press, 1960, pp. 152–168.
[64] M. TEN HOOPEN, A. DEN HERTOG, and H. A. REUVER, "Fluctuation in the excitability of nerve fibres, a model study," *Kybernetik*, Vol. 2 (1963), pp. 1–8.
[65] A. M. UTTLEY, "The classification of signals in the nervous system," *Electroenceph. Clin. Neurophysiol.*, Vol. 6 (1954), pp. 479–494.
[66] ———, "Variation of fibre density with distance—calculations using neurons in the striate and motor areas of cortex of cat," *Proc. Roy. Soc. Ser. B*, Vol. 144 (1955), pp. 229–240.
[67] ———, "A theory of the mechanism of learning based on the computation of conditional probabilities," *Proceedings of the First International Congress on Cybernetics*, Paris, Gauthier-Villars, 1956, pp. 830–856.
[68] ———, "The design of conditional probability computers," *Information and Control*, Vol. 2 (1959), pp. 1–24.
[69] M. A. VINCE, "Artificial acceleration of hatching in quail embryos," *Animal Behaviour*, Vol. 14 (1966), pp. 389–394.
[70] W. G. WALTER, *The Living Brain*, London, Duckworth, 1953, p. 4212.
[71] P. WEISS, "The problem of specificity in growth and development," *Yale J. Biol. Med.*, Vol. 19 (1947), pp. 235–278.
[72] S. WINOGRAD and J. D. COWAN, *Reliable Computation in the Presence of Noise*, Cambridge, Massachusetts Institute of Technology, 1963.