Cortical Activity Waves are the Physical Carriers of Memory and Thought

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Abstract— Growing and propagating waves of neural activity are the natural resonant modes of synaptic energy. In a layered geometry typifying the mammalian cortex, a time delay (T) in inter-layer signals effectively controls the temporal and spatial frequencies of the waves. As a function of T, two very different types of wave can grow from ubiquitous noise. One is coherent, and its resonant spatial frequency increases with increasing T. However, further increase eventually leads to a discontinuous increase in both wavelength and temporal frequency. The result is a region of T values wherein two waves grow simultaneously and interfere in random fashion. This remarkable duality, whose origin is in the phase relations of the amplified waves, leads us to propose that coherent waves are instrumental in the retrieval of memory and random waves embody original thought.

I INTRODUCTION

Mammalian brains are distinguished from those of reptiles primarily by their layered anatomy [1], which strongly implies that there is an evolutionary advantage in such a configuration. We propose here that this structure allows for a time delay within the connections between the layers; this delay has effective control over waves of neural activity, which are the resonant modes of cortical tissue and the electro-chemical energy stored within it. When there is sufficient stored energy these waves grow as they propagate to regions at distances from their point of origin considerably greater than the average axonal length, and at their saturated amplitude they involve the bulk of cells along their path.

The preceding statements are based on a numerical solution to the equations pioneered by Wilson and Cowan [2],[3] applied to a simple system: two filamentary layers connected through a spatially uniform time delay subject to variation on a slow time scale [4]. The Wilson-Cowan model focuses on fictitious "continuum elements" that act as energy reservoirs fed by synaptic flux from all other elements; the relationship between flux and stored energy resembles that of a "leaky capacitor". The energy is stored as the activity of the constituent cells, which are assumed to be of two types, excitatory (*e*) and inhibitory (*i*). The energy contained within each of the species is considered independently. The connections between elements are of four types, viz. *e-e*, *e-i*, *i-e*, and *i-i*, where the first index represents the afferent and the second index the efferent

species. Each afferent species has a characteristic connection probability that decreases with distance along the layers; the characteristic length over which this decrease occurs is the connection range. [2].

The neural continuum is subject to growing, propagating waves [5] [6]. If there is a slight increment in excitatory activity in one element, the *e-i* connections to adjoining elements cause increased inhibitory activity there, and return *i-e* connections suppress the excitatory surplus in the original element. As is often the case there is an overcorrection that results in oscillations. Meanwhile, *e-e* and *i-i* connections cause growth, and propagation through transmission to other elements. This process does not satisfy requirements for carriers of cognitive events, primarily because the parameters affecting wavelength and frequency are relatively fixed, subject to change only through synaptic modification [7].

The delay introduces control because of the phase relations necessary for wave amplification. Now an element with an excitatory surplus generates a wave as before, but that wave is split and a part propagates, with delay T, to the opposite layer. The relation between flux and activity in the second layer results in a phase shift very close to $\pi/2$. The same process occurs in the return to the original layer. Amplification can occur if and only if the total phase shift is an even multiple of π. Thus there is a multivalued relation between ω, the angular temporal frequency of the favored wave, and the delay

$$\omega T = (n + \tfrac{1}{2})\pi \qquad n = 0,1..$$

(1)

In any given situation the value of integer n selected is the one that leads to the greatest growth, which is initially exponential [4],[8]. Within some distinct ranges of delay the rapidity of this growth leads to clear dominance of the selected mode. However as T increases a new mode appears with comparable growth rate and discontinuously higher frequency. For activity waves, the wavelength and propagation speed are both increasing functions of frequency and thus they also jump. The two waves coexist with different speeds of propagation and at saturation they occupy different regions of space; in between, the pattern of activity is highly irregular, increasingly so over time. This condition persists until T reaches a value at which the new mode dominates.

II THE WILSON-COWAN EQUATIONS

In our model the dependent variables (four in all) are the respective active fractions A_{sl} (x,t) $s= e,i, l = 0,1$, at time t,

of the different species in the two layers, within an element centered at position x.

$$\frac{\partial}{\partial t} A_{sl} + A_{sl} = S(N_{sl})$$

(2)

Here **N** is the net synaptic flux into the element from all other elements, weighted by the decrease of connection probabilities with distance, and the time delay in inter-layer signals.

$$N_{sl}(x,t) = \sum_u \sum_m \int_{-\infty}^{\infty} dX F B_{us,ml} A_{um}(X, t - T_{ml}) e^{\frac{-|X-x|}{\sigma_{us,ml}}}$$

(3)

The connection coefficients $B_{us,ml}$ are proportional to the afferent connection probability of a cell of species u and layer m to the species and layer in question when the cells are at the same lateral position (B is negative when $u = i$). The exponential represents the decrease of connection probability with lateral distance from X, the position of the afferent element, to the element at x; the connection range $\sigma_{us,ml}$ is assumed to depend only on the afferent species. In this calculation all inter-layer connection parameters are assumed to be the same in both directions and the connections within the layers are identical. Delay T_{ml} applies only if m and l refer to opposite layers, when it equals T. F represents the synaptic energy conveyed by the typical synapse, and is thus an attention parameter. T and F are held constant during experiments. The sums are over both species and layers, and the integral is over all space.

The sigmoid function S is a flux limiter, necessary because the total stored energy cannot increase when all the cells are active or decrease when none are. It is assumed that there is an equilibrium activity level, taken to be ½ for both species and layers; the active fractions thus lie between –½ and +½. The functional form used for S has a value of zero and slope of unity when the synaptic flux is zero. The second term in (2) represents a decay towards equilibrium with time constant unity. The spatial unit throughout is the same-layer inhibitory connection range

III NUMERICAL RESULTS

Fig. 1 shows the results for two values of delay. (For clarity only positive wave amplitudes are shown. Negative amplitudes are slightly larger because of the neural refractory period [2]). In our code lateral space is divided into 512 segments. At $t = 0$, a δ-function impulse of amplitude 0.0001 is assumed to be added to the excitatory active fraction in one of the layers at $x = 0$; the first time step is executed analytically to seed all the points. To advance in time the equations in (2) (2048 amplitudes) are solved simultaneously using an adaptive Runge-Kutta-Fehlberg *(RKF)* method, which has a built-in test of convergence. At each function evaluation the integral in (3) is performed using a cubic spline integrated analytically (use of discrete methods leads to spurious spectral lines).
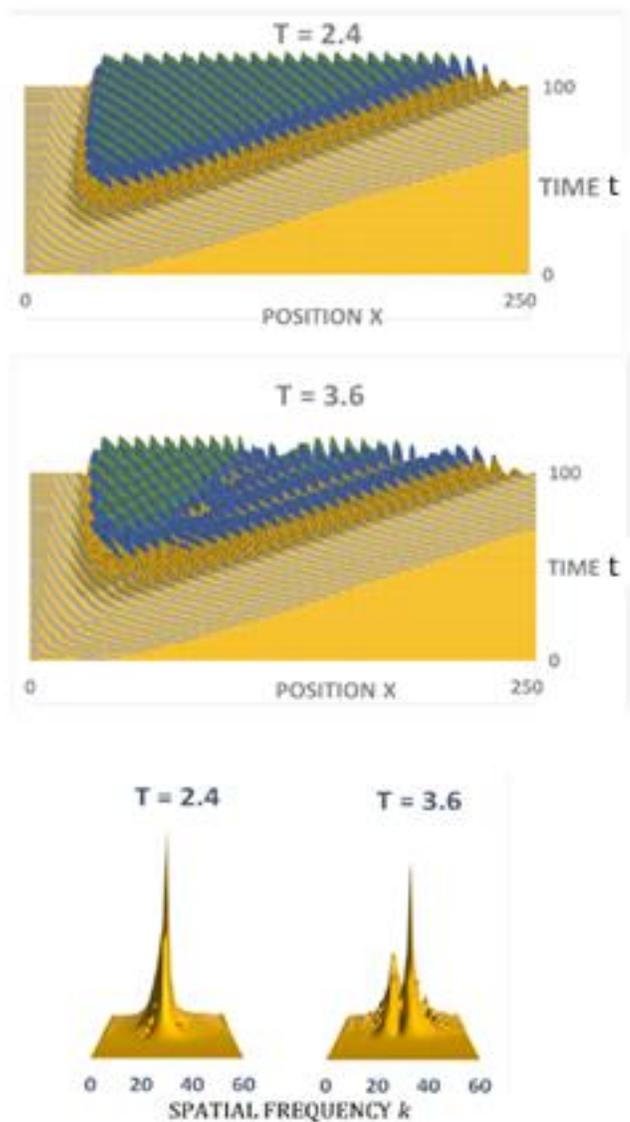
Figure 1. (top) The waves generated, for two values of delay T, by a small increment (0.0001) in the excited fraction of e-cells in an element at position 0 in layer 0 at time 0. Spatial units are in terms of the same-layer inhibitory range, and time units are decay periods. Only the positive amplitudes in the stimulated layer are shown. For $T = 2.4$, a single wave grows exponentially until saturation, caused by the flux limit; the maximum amplitude is about 0.4. For $T = 3.6$ two competing waves with different wavelengths are launched, occupying different regions of space. Activity in intermediate regions is irregular. (bot.) The spectral intensities of the waves as a function of wave number k ($k = 256/\lambda$ where λ is the wavelength) and time (into the diagram). At $T = 2.4$ the spectrum has a single narrow peak, indicating coherence. At $T = 3.6$ the energy is distributed between two peaks; at late times other waves of shorter wavelength are excited by non-linear interference. The connection parameters in the same layer are: $B_{ee} = 3.5$, $B_{ei} = 5.9$, $B_{ie} = -4.8$, $B_{ii} = -2.5$, $\sigma_e = 1.25$, $\sigma_i = 1.0$; for opposite layers $B_{ee} = 3.0$, $B_{ei} = 1.1$, $B_{ie} = B_{ii} = -1.0$, $\sigma_e = \sigma_{ii} = 0.6$. The attention factor $F = 0.45$.

To account for the delay, temporary files are used to store the amplitudes until needed. These are written at fixed time intervals (0.1 decay periods); intermediate values necessary for *RKF* are obtained by linear interpolation. To avoid end effects the calculation is terminated when a detectable signal reaches the final point. After each step a fast Fourier transform (*fft*) is performed; the relationship
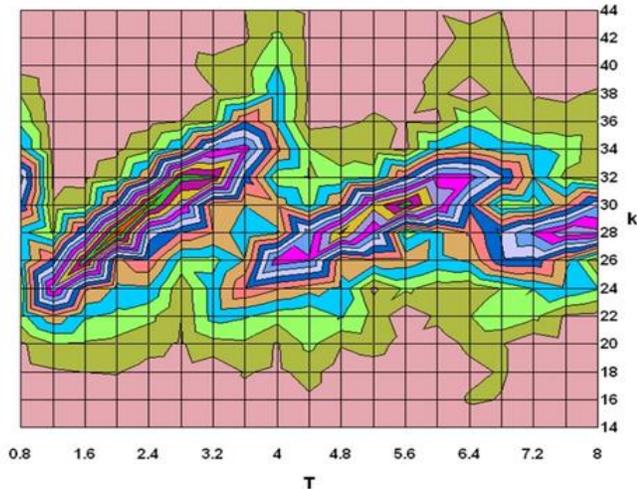
Figure 2. This is a contour plot of the spectral intensity at a fixed time after stimulus (80 decay periods), as a function of delay, in a series of experiments during each of which delay is held constant. The other parameters are the same as in the preceding figures.

between spectral variable k and wavelength λ is $k\lambda = 256$.

The regularity of the wave at $T = 2.4$ and the narrowness of the spectral peak indicate that it has a high degree of coherence. **Cells in elements one wavelength apart are active and inactive in lockstep, in spite of the random location of the initial noise**. At $T = 3.6$, two waves are generated; they have different growth rates and, because of their different wavelengths, they have different propagation velocities and occupy different spatial regions. In the intermediate space there is irregular activity.

Figs. 2 and 3 show some of the effects of delay

IV CONCLUSION

In constructing Figs. 2 and 3 we conceive of a succession of cognitive events [10] with the delay increasing between them. We note from Fig. 2 that at values of T between about 1.2 and 2.8 the spectrum is single peaked, with the favored wavelength decreasing with successive events (it remains greater than any of the connection ranges) and the wave form is regular at least until $T = 2.8$ (Fig 3). The wave organizes the neurons into subsets, and changing the delay regroups them. This process can be likened to a memory search, since repetition of a value of T results (more or less perfectly) in the reconstitution of its associated groups (cells one wavelength apart).

We do not take sides here on the theories of memory storage nor do we speculate on how the waves relate to
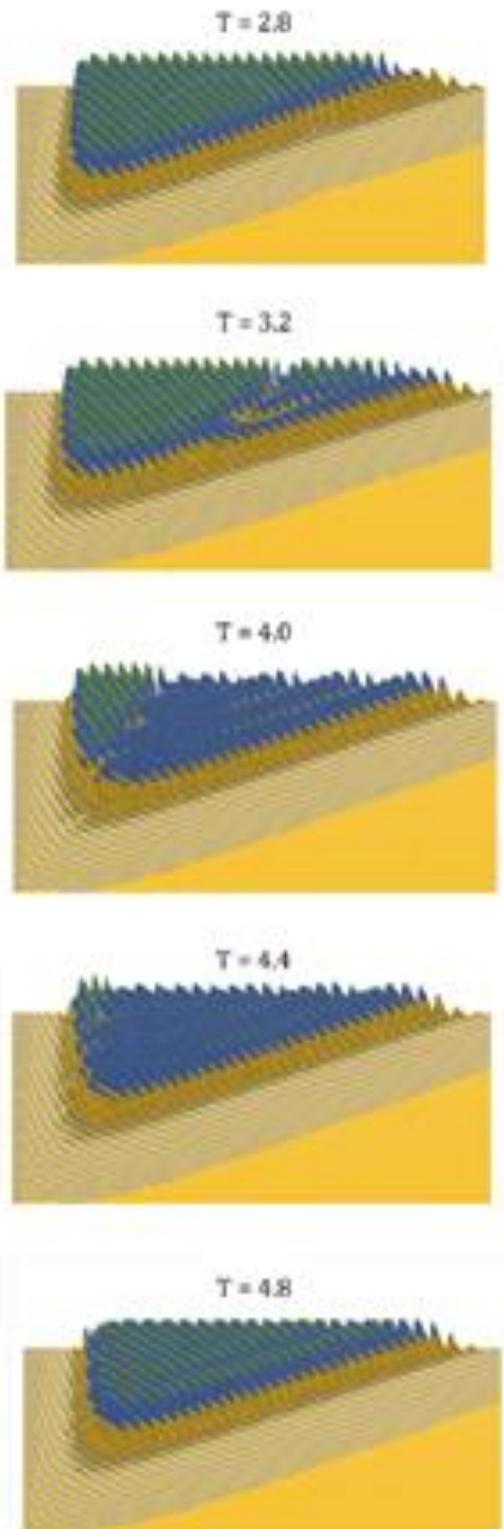
Figure 3. The wave forms generated at several values of delay T. The other parameters are the same as in the previous figures.

semantic content. But activity waves are the resonant ~generated~ modes associated with our simple model, and insofar as it approximates the real cortex, they must be taken into account in these theories.

Above $T = 2.8$ a second wave begins to grow and to occupy regions far from the origin. Referring to Fig. 3, we note that its growth rate is smaller than that of the original wave; by $T = 4.4$ this situation is reversed and the second wave dominates, occupying most of space. At $T = 4.8$ wave is regular again, and in fact we learn   from Fig. 2 that the wavelength is about the same as for $T = 2.4$ (however the growth is not as rapid).

Within this transition the wave forms are very irregular, most notably in the space between the waves. Also the spectra are considerably more diffuse and spead to larger values of wave number, indicating the presence of small-scale waves generated by non-linear interference. The random location of the initial stimulus now plays a role because the location of the irregular wave pattern is fixed only with respect to this origin. Therefore there are numerous different possible neural firing patterns in such cases. If each active subset of individual cortical cells represents a distinct thought, there are about $10^{6\ billion}$ such possibilities for about 20 billion cortical cells; in the spirit of the continuum analysis wherein cells are grouped into elements there are considerably fewer possibilities, but still there are very many.

This theory does not try describe how these patterns are "decoded" into a mixture of the semantic content of different wave-memories and memories of previous thoughts. If we do assume that such a translation exists, the wave forms of Fig. 3 may give a hint as to how the delay controls the creative proces, if we allow it to change randomly, within the transition region, in successive moments of perception [10]. All mammals are subject to cortical activity waves, but only humans can associate language (words, music, fine art, dance, algebra, etc.) with the waves, and thus can have memories of our thoughts.

With respect to the validity of our assumptions we note that the six-layered structure of the human cortex opens up the possibility of numerous delays and consequent rhythms. The origin of the various electroencephalographic frequency bands [11] is not yet explained [12]. Clearly the present theory speaks directly to that problem.

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