Neuronal Avalanches

Ethan W. Brown

Physics 563 Term Essay, Nigel Goldenfeld, Spring 2010

Abstract

It has recently been suggested that, in order to provide the richness of behavior which is seen empirically, the brain must operate in a critical regime between a phase of order and disorder [1]. In particular, such critical behavior is exhibited in what is termed neuronal avalanches. These cascades of synaptic activity have been seen both in vitro and in vivo [6]. Several models have been put forth to explain this apparently critical phenomena, ranging from simple dynamical models of neural networks to full fluctuation field theories, though the specific mechanism remains unclear. Despite the reproducibility of this behavior, its connection to high-order processes is yet unknown, though some have linked neuronal avalanches to large scale brain features such as information storage [2, 4], synaptic stability [3], even epileptic shock [1]. In hopes of providing a larger context for this pervasive neural behavior, we examine the aforementioned experimental findings and compare various theoretical models which predict them.

The brain is often seen as a complex, non-linear system in which large-scale dynamics emerge from small-scale interactions. Local functional groups of neurons are often easier to characterize through physiological studies, explaining relatively isolated brain functions. However, any whole brain process, e.g. memory and learning [5], requires spatially distant groups to work in concert. This functional interplay between cortical segregation and integration is not well understood [1]. Over the past 25 years, the brain's inherent ability to perform within this dichotomy of scale has increasingly been seen as operation on the brink of criticality, i.e. at a point where interactions show no characteristic scale, and fluctuations exhibit power-law behavior [1]. In a critical system, isolated perturbations by either external or internal stimuli can quickly cascade throughout the system, resulting in complex responses both locally and globally. Through this critical avalanching, the brain may be afforded the speed and flexibility required to react to novel input [2], while at the same time allowing for long-lasting, long-range interactions [3, 4]. In the proceeding, we attempt to characterize this critical behavior, citing specific experimental evidence and models which predict it.

In the brain criticality is seemingly spontaneous, in contrast with typical second order phase transitions where an external parameter must be tuned near a critical value in order to achieve scale-invariance. Such self-organized criticality (SOC) was first explained by Per Bak [3] in a seminal paper describing the ubiquitous power-law dependence of power spectra on frequency, $P(f) \sim f^{-\beta}$, in dynamical systems. Such behavior exists in systems ranging from earthquakes [13] to forest fires [14]. In these systems, individual units are perturbed past some threshold whereupon they relax, dissipating energy back to the system. This dissipation can in turn cause neighboring (or distal) units to exceed their threshold, thus causing a cascade of excitation and dissipation. What is truly remarkable about this cascade is that it lacks a characteristic size, which leads directly to a lack of characteristic lifetime. Instead, these observables follow a power law. For example, under perturbation, a distribution of cascade size s could be given by $D(s) \sim s^{-\tau}$. If the perturbation grows within the cascade with exponent γ, then the cascade's lifetime goes as, $t^\gamma \sim \frac{s}{t}$. This in turn gives a distribution lifetimes $D(t) = \frac{s}{t} D(s(t)) \frac{ds}{dt} \sim t^{-(\gamma+1)\tau+2\tau} \equiv t^{-a}$. Such a distribution leads to a frequency spectrum

$$S(\omega) = \int dt \frac{tD(t)}{1+(wt)^2} \sim \omega^{-2+a} \tag{1}$$

hence the term "$1/f^\beta$" noise [3].

Representing this activity through cellular automata, Bak demonstrates that without external tuning, the system drives towards a critical state. At this point perturbative events cause the spacial and temporal distributions of minimally stable states (domains) to exhibit a scale-invariant power-law dependence on the size and lifetime of such states, respectively. Because of this lack of characteristic scale, the correlation length between sites diverges and the number of degrees of freedom is drastically reduced. More simplistically, a self-organized critical system may be seen as "a driven, dissipative system consisting of a (1) a medium which has (2) disturbances propagating through it, causing (3) modification of the medium such that eventually (4) the medium is in a critical state, and (5) the medium is modified no more" [15]. From the above description, one can see directly how the brain may be thought of as a SOC system. Analogous to a single site in the cellular automata lattice, individual neurons integrate synaptic input from up to 10^4 synaptic connections [5]. When this input reaches some threshold voltage, the neuron sends a spike down its axon, redistributing pre-synaptic
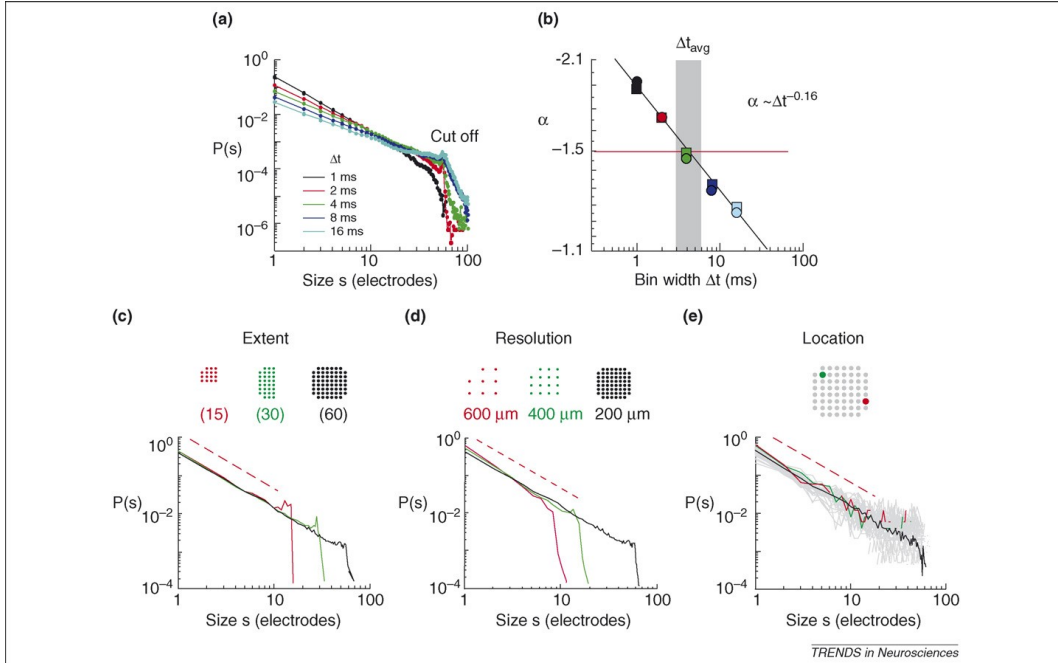
Figure 1: Beggs and Plenz original avalanche size data. (a) Distribution of avalanche sizes for various bin sizes, Δt. (b) Power law law exponent α varies with Δt. Note that t_{avg} gives $alpha \sim -1.5$. (c,d) Data rescaled so that Δt is the average interelectrode event interval, thus collapsing on the power law exponent $alpha \sim -1.5$. Note that this result in invariant of both spatial scale and interelectrode distance. (e) Avalanches initiated at a single site show the same power law behavior with $alpha \sim -1.5$. [7]

activity to the rest of the system. Furthermore, the propagating disturbances, termed *neuronal avalanches*, have been measured both in vivo and in vitro, most notably in 2003 by Beggs and Plenz [6]. Having seen evidence of self-organized criticality in other natural systems, they hypothesized that the complex network of cortical neurons could exhibit similar behavior.

In order to test this hypothesis, Beggs and Plenz studied the propagation of spontaneous neuronal activity in mature rat cortex slices and cultures on 60-channel multielectrode arrays. During their trials, each electrode was continuously sampled, binning excitations which surpass a preordained threshold local field potential (LFP) every Δt. The size of a neuronal avalanche is then defined as the number of binned events within this interval, i.e. the number of involved electrodes. The duration of an avalanche was determined by the length of time between two Δt intervals during which no activity was measured.

Defined this way, Beggs and Plenz record neuronal avalanches of a variety of sizes and lengths. A log-log plot of the distribution of avalanche sizes reveals a simple power law, $P(n) \sim n^{\alpha}$, where n is the size of the avalanche and alpha is the slope of the plot, Fig. 1. At first glance, alpha is different for each temporal bin size, Δt. However, Beggs and Plenz are able to remove avalanche size bias by allowing Δt to be precisely the average interval between firing events. After doing so, their data collapses into a single power law with $\alpha = -3/2$. The cutoff for this linear log-log relationship is consistently solely dependent on the number of electrodes present. Similarly a log-log

3

plot of the distribution of avalanche lengths reveals a temporal power law relationship with exponent $\alpha = -2$ and exponential cutoff. Both these results stood up to different variations of the experiment, including various threshold potentials and system sizes [6].

What Beggs and Plenz found was a wholly new mode of neural activity. Up until this point, activity in the cortex was seen as limited to oscillations, waves and synchrony [16]. The neuronal avalanches measured by Beggs and Plenz showed little spatial confinement, making them distinct from waves which propagate across neighboring synapses. Additionally, the avalanches lacked regular temporal structure, an inherent property of oscillations.

Phase synchronization among cortical oscillations have been shown to link neurons together into functional groups which can then respond collectively to stimuli [16]. These neuronal assemblies composed of spatially distant elements are then locked together by a common phase signal, thus allowing for long-range communication across the cortex. However, this oscillatory activity alone has failed to completely describe the particular long-range correlations needed for mammalian consciousness [16]. Neuronal avalanches may be able to complete this picture. As a direct result of residing in the critical regime, correlation lengths diverge. Thus, the only limiting factor in avalanche size is the size of the system. This implies that neuronal avalanches originating at one site in the cortex may access information stored at any other site. In fact, the avalanches measured by Beggs and Plenz often exceeded the width of a single cortical column, $\sim 1.6mm$, allowing for the possibility of avalanche mediated cross-cortical communication [6].

As mentioned earlier, critical and power law behaviors are naturally ubiquitous. As such, there exists a large body of theoretical work which attempts to explain these phenomena. We will now briefly discuss three such models that apply directly to criticality and avalanches.

Probably the simplest account of power law behavior in avalanches is given by the critical branching process. In these models, a single active site relaxes with probability p, dissipating energy to two other sites and making them active. These newly activated sites then relax with probability p or stop the chain with probability $1 - p$ [7]. This process continues until each chain has been terminated, Fig. 2. In order to map this process to a SOC system, Zapperi et. al. created a model which allows for energy dissipation through open boundary conditions [8]. In this way, the system will attempt to balance energy exiting by dissipation with the energy entering by perturbation. Following their proof, suppose only a finite number n of generations are allowed, and let γ_n be the number of remaining active sites in the nth generation and N be the total number of affected sites. Then,

$$p(1 + t) = p(t) + \frac{1 - \gamma_n}{N} \tag{2}$$

so that if $\gamma_n > 0$, p decreases (since more energy is leaving than entering) and if $\gamma_n = 0$, p increases (since more energy is entering than leaving). Using this recursion relation in a computer model, they find a critical value for p, $p_c = 1/2$, about which the system oscillates. Note that in branching processes, another measure of p is the branching parameter, σ, which is the ratio of ancestral states to the number of descendant states. Thus, $p = 1/2$ corresponds to $\sigma = 1$. At this value, they find the avalanche size distribution to obey a power law, $D(s) \sim s^{-\tau}$ with $\tau = 3/2$.
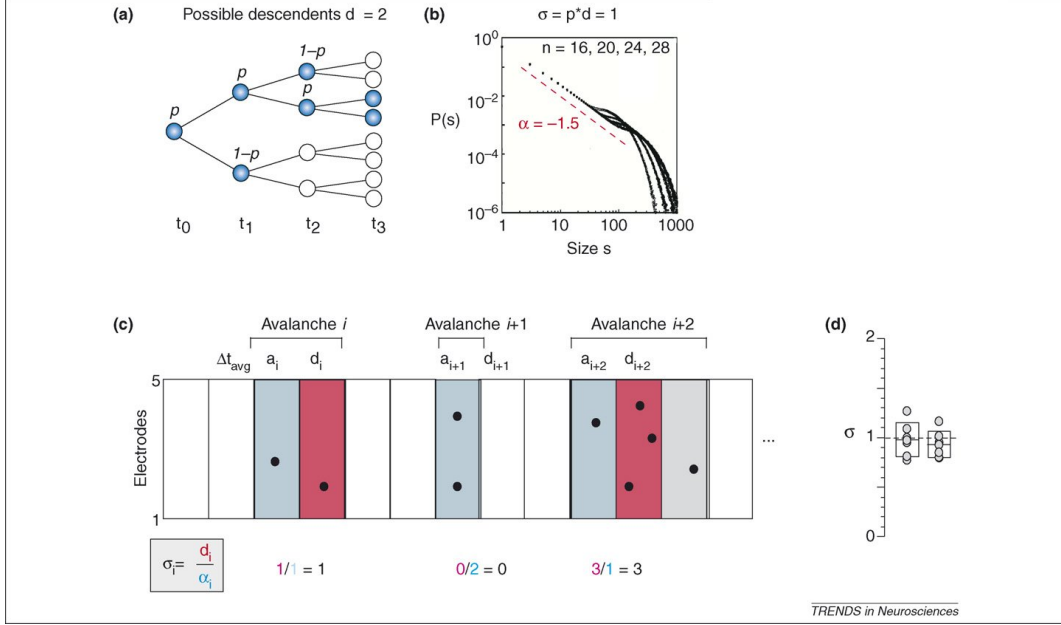
Figure 2: Neuronal avalanches as branching processes. (a) A simple branching process where at each t_i, the active site has probability p of continued propagation. (b) Beggs and Plenz critical branching process model shows good convergence to a power law relationship with $alpha = -1.5$. These results persist for several system sizes. (c) Calculation of the branching parameter σ as the ratio of descendant events to ancestral within each avalanche. (d) Beggs and Plenz experimental measurement of the branching parameter is approximately 1. [7]

Using a mean field for γ_n, $\langle \gamma_n \rangle = (2p)^n$ they are able to analytically derive the distribution of avalanches sizes,

$$P_n(s, p) = \frac{\sqrt{2(1-p)/\pi p}}{s^{3/2}} \exp\left(-s/s_c(p)\right) \tag{3}$$

where $s_c(p) = -2/ln(4p(1-p))$ is the cutoff size. It is clear then that as $p \to 1/2$, $s_c(p) \to \infty$, implying that $p_c = 1/2$ and $\tau = 3/2$ as was decided numerically. Finally, a similar process may be used to compute the lifetime distribution, $D(t) \sim t^{-\alpha}$, giving $\alpha = 2$.

Clearly these exponents match the power law behavior seen by Beggs and Plenz. Noting that one group neuronal activation giving rise to another may be seen as a branching process, Beggs and Plenz measured the branching ratio throughout their experiment. To do so, they approximated σ as the ratio of firing events in one time bin to the next, giving them $\sigma = 0.90 \pm 0.19$ for all avalanches and $\sigma = 1.04 \pm 0.19$ for avalanches initiated by a single ancestor, Fig. 2.

Though these results make it seem reasonable to assume neuronal avalanches are critical branching processes, there exist a few caveats that seem to limit this model's tractability. First, if the brain really is self-organized critical, the tuning of an external parameter such as p or σ would be unnecessary. Though it should be noted that other experiments have seen this $\tau = 3/2$ power law to depend on dopamine concentrations or inhibition/excitation levels, implying that specific parameters may need to be appropriately tuned for criticality [9]. Second, branching processes assume that descendants
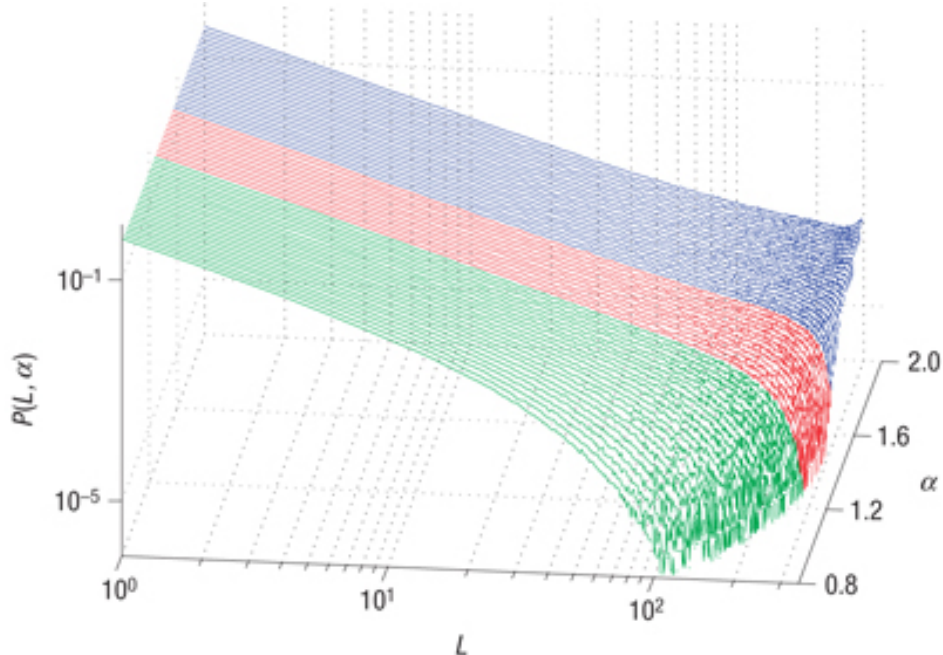
5

Figure 3: Distribution of avalanche sizes plotted for several coupling strengths, α. The critical range (shown in red) is for $1.3 < \alpha < 1.6$. Below this range no events span the system, while above this range a substantial fraction do, implying a mechanism for epileptic shock. [11]

have no knowledge of their ancestors, thus making each generation independent of the previous one. This seems unlikely in the cortex where synaptic connections not only have inherent latency, but also varying plasticity. Thus connections between neurons have at least some sort of memory [7]. Furthermore, this variance of synaptic strength seen in the cortex argues against the possibility of uniform branching probabilities. Finally, Zapperi et. al. were only able to produce analytic results in the thermodynamic limit, i.e. $N \to \infty$, but the cortex, though consisting of many neurons, is by no means infinite in extent.

Some time after this, Eurich, Hermann, and Ernst put forth a model based on the Olami-Feder-Christensen stick-slip model which accounted for the finite size behavior of avalanches, but still required the tuning of an external parameter to achieve power law behavior [10]. In an effort to account for this additional caveat, Levina, Hermann, and Geisel create a model which achieves criticality by assuming more physiological synapses [11]. In particular, synapses in their model are activity-dependent, thus coupling strengths between neurons weakens with increased activation. This is in line with many findings [9] which point to depressive synapses. The following is a direct account of this model. Consider N integrate and fire neurons, each with membrane potential $h_i(t)$ and threshold potential θ. At time t, these neurons receive external input I^{ext} at site $\xi_\tau(t) = i$ at a rate τ. If at any point $h_i(t) > \theta$, the neuron waits a time τ_d and dissipates (spikes), sending input to all postsynaptic neurons with coupling strength

J_{ij}. This leads to the following relation for the membrane potential,

$$\dot{h_i}(t) = \delta_{i,\xi_\tau(t)} I^{ext} + \frac{1}{N} \sum_{j=1}^{N} u J_{ij} \delta(t - t_{sp}^j - \tau_d) \tag{4}$$

where t_{sp}^j is the time at which neuron j last spiked. To enforce depressive synapses, J_{ij} obeys the following dynamics,

$$\dot{J_{ij}} = \frac{1}{\tau_J}(\frac{\alpha}{u} - J_{ij}) - u J_{ij} \delta(t - t_{sp}^j) \tag{5}$$

Thus for each spike that arrives at a synapse, the strength of that synaptic connection is diminished by a fraction u, physiologically implying a depletion of neurotransmitter. During periods of no activity, the coupling constant recovers at a rate $\tau_J = \tau\nu N$ to its maximum value α/u, where $1 < \nu \ll N$.

In order to inspect the behavior of this system, the mean value $\langle J_{ij} \rangle$ with respect to the interspike interval Δ^{isi} is found giving a uniform synaptic strength $\alpha_0 = u\langle J_{ij} \rangle$. It is important to note that this averaging does not skew the dynamical result since the coupling constant does not exhibit power-law behavior at criticality, Fig. 3. Intuitively, for short interspike intervals, the synapses are more frequently depressed leading to a smaller average coupling strength. On the other hand, for large synaptic strengths, avalanches occur more readily causing shorter interspike intervals. Analytically, this interplay can be seen in self-consistency equations.

$$\langle J_{ij} \rangle = \langle J_{ij} \rangle(\langle \Delta^{isi} \rangle) \text{ and } \langle \Delta^{isi} \rangle = \langle \Delta^{isi} \rangle(\langle J_{ij} \rangle) \tag{6}$$

whose mean field solution gives the behavior of the system. This information is then sufficient to determine the distribution of avalanche sizes, and thus if applicable, extract power-law exponents.

Levina et. al. found that for a range of synaptic strengths α, the interdependence of network activity and coupling results in a self-organized drifting toward critical avalanching dynamics, Fig. 3. Specifically, in the thermodynamic limit ($N \to \infty$), the network becomes critical, exhibiting power-law behavior for $\alpha \geq 1$. This range of synaptic strengths give a range of power-law exponents which includes the Beggs and Plenz value of $3/2$. Levina et. al. were able to numerically extend these results to include networks with that were not fully connected and included biologically realistic leak currents. However, analytic confirmation of these extensions was deemed intractable [11].

The above models provide analytic calculations solely for a uniformly all-to-all connected network. Further, other measurable quantities, e.g. correlation functions, are not given explicity analytic form. This makes testing these results difficult. In a effort to extend beyond this restriction, Buice and Cowan, use the methods of stochastic field theory applied to non-equilibrium statistical mechanics in order to generate a field-theoretic "effective spike" model [5]. In it a continuous spike field $\phi(x,t)$ is used in place individual spike records. Such statistical mechanical continuation is seemingly admissible simply due to the immense number of synaptic connections made in the cortex.

In their model, a site configuration is given by the number of effective spikes n_i emitted by neuron i. Neurons fire according to a rate function $f(s)$, which depends

on input s. This input is (as in Levina et. al. [11]) the sum of external input I^{ext} and recurrent input from other neurons $\sum_j w_{ij} n_j$, where w_{ij} is the relative synaptic strength between neuron i and j. Finally, a single spike's effectiveness decays as $1/\alpha$, which may be translated as the contribution of inhibition. In this way, n_i describes the total number of spikes initiated at neuron i which are still affecting the system's dynamics. Thus, $n_i \rightarrow n_{i-1}$ represents a spike from neuron i losing effectiveness. Likewise, $n_i \rightarrow n_{i-1}$ represents neuron i spiking.

With the assumption that this is a Markov process, one arrives upon a dynamical master equation

$$\frac{dP(\vec{n}, t)}{dt} = \sum_i \alpha(n_i + 1)P(\vec{n_{i+}}, t) - \alpha(n_i)P(\vec{n}, t) + f(\sum_j w_{ij} n_j + I^{ext})[P(\vec{n_{i-}}, t) - P(\vec{n}, t)]$$
(7)

where $P(\vec{n}, t)$ is the probability of the system having spike configuration $\vec{n}$ at time t. Note also, the use of the notation $\vec{n_{i\pm}}$ denoting the configuration $\vec{n}$ where the ith component is $n_i \pm 1$.

We now briefly overview Buice and Cowans' analytic solution of their model [5]. In order to create the corresponding field-theoretic model, they build up states $| \vec{n} \rangle$ using defined creation and annihilation operators. Next, these states are transformed into field variables, $\phi(x, t)$ and $\tilde{\phi}(x, t)$ using a coherent state representation. Here, $\phi(x, t)$ describes $\vec{n}$ while $\tilde{\phi}(x, t)$ describes the systems response to perturbations. These new fields are related back to the original configuration as follows

$$\langle \prod_i n(x_i, t_i) \rangle = \langle \prod_i [\tilde{\phi}(x_i, t_i)\phi(x_i, t_i) + \phi(x_i, t_i)] \rangle$$
(8)

It is often more convenient to characterize a dynamical system by a partition sum Z, where the sum is over all realizations of the field weighted by an appropriate action. Thus they produce a generating functional $Z[J, \tilde{J}]$ as a path integral over field variables.

$$Z[J, \tilde{J}] = \int \mathcal{D}\phi \mathcal{D}\tilde{\phi} \exp\left(-S[\phi, \tilde{\phi}] + J \cdot \tilde{\phi} + \tilde{J} \cdot \phi\right)$$
(9)

where they have introduced external currents

$$\tilde{J} \cdot \phi = \int d^d x dt \tilde{J}(x, t)\phi(x, t)$$
(10)

The weighting action is given by

$$S[\phi, \tilde{\phi}] = \int_{-\infty}^{\infty} d^d x \left(\int_0^t dt \tilde{\phi}\partial_t \phi + \alpha\tilde{\phi}\phi - \tilde{\phi}f(w \star [\tilde{\phi}\phi + \phi] + I)\right) - \int_{-\infty}^{\infty} d^d x \overline{n}(x)\tilde{\phi}(x, 0)$$
(11)

where $\star$ denotes a convolution and $\overline{n}(x)$ is an initial state operator. In this particular model, $\overline{n}(x)$ corresponds to a configuration where each neuron has an Poisson distribution of spikes.

The generating functional contains all statistical mechanical information about the system, as a partition function does in non-field theories. Thus, in order to gain insight into the dynamics of the system, one must only calculate $Z[J, \tilde{J}]$. Clearly, this is not always a straight-forward task. The usual way of proceeding is a simple perturbative expansion of the action with Feynman diagrams. However, Buice and Cowan decide

to exploit the convenience of a loop expansion via mean field theory and the method of steepest descents.

In order to determine when this expansion is valid, a limiting condition is developed analogous to the Ginsberg criterion of equilibrium statistical mechanics with correlation length replaced by diffusion length. Denoting w_n as the nth moment of the distribution $w(x)$, the criterion is explicitly,

$$(\frac{w_2}{w_0})^2 \gg \frac{|f''|w_0 A[f', w_4, ...]}{f'} l_d^{4-d}$$

where

$$l_d^2 = \frac{f' w_2}{2(\alpha - f' w_0)}$$

(12)

is the diffusion length, i.e. the length which single spike effects will propagate before decaying [5]. As this length approaches the physical size of the system, the loop correction begins to fail since small spike fluctuations may then have a non-negligible effect. Additionally one can see mean field approximations are valid only for $d > 4$. Clearly in most finite physical systems, $d = 2$ or 3. However, if one considers the multiplicity and range of cortical interactions, d can be taken to be much larger. Nevertheless, mean field theory is traditionally only consistent far away from the critical point. In order to determine dynamics within the critical regime, Buice and Cowan turn to renormalization group.

By making the following transformation of fields

$$\tilde{\phi}(x, t) \rightarrow \sqrt{\frac{2f'}{w_0 |f''|}} \tilde{\phi}(x, t)$$

$$\phi(x, t) \rightarrow (\sqrt{\frac{2f'}{w_0 |f''|}})^{-1} \phi(x, t)$$

(13)

where w_0 is the zeroth moment of the synaptic weight distribution, the action may be written

$$S[\phi, \tilde{\phi}] = \int dt d^d x [\tilde{\phi} \partial_t \phi - D\tilde{\phi} \bigtriangledown^2 \phi + \mu \tilde{\phi} \phi + g(\phi^2 \tilde{\phi} - \tilde{\phi}^2 \phi)$$

(14)

where D, μ, and g are defined renormalized parameters representing diffusion, decay, and coupling constants, respectively. Note that all terms whose coefficients scale to 0 at large system sizes have been dropped.

This is precisely the action seen in Reggeon field theory, and hence in the same universality class as directed percolation (18). Percolation can generally be seen as fluid flowing through a semi-permeable membrane. A directed percolation system goes through a phase transition from a non-percolating (impermeable) state to a percolating (permeable) state. In the same way, Buice and Cowan predict the cortex to undergo a phase transition from a quiescent state to an active fluctuating state through $\mu = 0$. In this system, $\mu_0 = \alpha - f' w_0$ implying that the transition occurs precisely at the point wherein the propensity of a neuron to fire matches its spike decay rate. This may also be interpreted as the point at which inhibition, α, balances excitation. A directed percolation is characterized by the critical exponents β, β', ν, and z. These give the

power law behavior of

$$\langle \phi(x,t) \rangle \sim \mu^{\beta}$$
$$\xi_s \sim \mu^{\nu}$$
$$\xi_t \sim \mu^{\nu z}$$
$$P_{\infty} \sim \mu^{\beta'}$$

(15)

which are the spike field expectation value, spatial correlation length, temporal correlation length, and probability that a site will belong to a time-spanning cluster, respectively. It is known that for $d \leq 4$, these values are not analytically solvable [12]. However, for $d > 4$, we may use their mean field values, $\beta = 1$, $\nu = 1/2$, and $z = 2$. Under directed percolation, the distribution of avalanche sizes scales as $D(s) = s^{1-\tau} g(\mu s^{\sigma})$ with

$$\tau = 2 + \frac{\beta}{(d+z)\nu - \beta} = \frac{5}{2}$$
$$\sigma = \frac{1}{(d+z)\nu - \beta} = \frac{1}{2}$$

(16)

where the mean field values have been inserted and $d = d_c = 4$. Thus the Beggs and Plenz result is recovered, $D(s) \sim s^{-3/2}$ [5].

Buice and Cowan arrived upon their results with few qualifying assumptions, however, it is important to understand what they were. First and foremost, they assume cortical activity is a Markov process. This implies that each successive generation of spike configurations has no knowledge of the past. As noted earlier for critical branching processes, this assertion is not necessarily physiological well-founded. Second, even though they relax the condition of all-to-all synaptic connections, they reimpose the condition of uniform synaptic strength. Again, as noted earlier, this discounts the possibility of recurrent synaptic chains. Furthermore, the critical state requires tuning the magnitude of intercortical interactions, and thus is not by definition self-organized. Though, as they rightly point out, self-organized criticality has been speculated to be tuned directed percolation where the tuning parameter is just difficult to uncover [5]. Or it may be possible that a process as in Levina et. al. tunes this parameter automatically. (It is interesting to note that it is no coincidence that these assumptions are shared with our first model. In fact, for $d > 4$, directed percolation displays precisely the dynamical behavior of a critical branching process [5].)

Despite these possible points of contention, what is most intriguing is the ability to place this problem in a universality class. In doing so, Buice and Cowan have shown how it is possible that a host of disparate models may describe the same critical phenomena. They intuitively point out that a model's biological resemblance says "more about the evolutionary path by which that system was realized than about the actual function" [5]. Thus in uncovering purpose, finding a physiologically accurate model becomes secondary to finding the appropriate universality class.

Because of this, it is worth understanding how different physical parameters change a model's universality. For example, the introduction of a post-spike refractory state moves their model from directed percolation to dynamic isotropic percolation. Furthermore, it has been shown that spatial and temporal disorder separately can alter universality, though spatiotemporally quenched disorder (as found in the cortex) does not [5].

Each of the above models produces the experimentally discovered power-law in neuronal avalanches. However, deciding which one is more biologically accurate seems

almost a matter of taste rather than physicality. Certain qualifying assumptions most likely place each into a universality class. It is thus now back on the experimentalist to idenitify the precise nonequilibrium behavior and universality. With this information, the theorist can know which class of models describe neural dynamics. In this way, we hope to better understand not only the underlying mechanism, but also the function of criticality in the brain.

References

[1] D. R. Chialvo, P. Balenzuela, D. F. 2008, *arXiv:0804.0032v1*

[2] Tononi, G. 2004, *BMC Neurosci.*, 5

[3] P. Bak, C. Tang, K. W. 1987, *Phys. Rev. Lett.*, 59

[4] Haldeman, C. & Beggs, J. M. 2005, *Phys. Rev. Lett.*, 94

[5] Buice, M. A. & Cowan, J. D. 2007, *Phys. Rev. E*, 75

[6] Beggs, J. M. & Plenz, D. 2003, *J. Neurosci.*, 23

[7] Plenz, D. & Thiagarajan, T. C. 2007, *Trends in Neuroscience*, 30

[8] S. Zapperi, K. B. Lauritsen, H. E. S. 1995, *arXiv:cond-mat/9509161v1*

[9] Stewart, C. V. & Plenz, D. 2006, *J. Neurosci.*, 26

[10] C. W. Eurich, J. M. Herrmann, U. A. E. 2002, *Phys. Rev. E*, 66

[11] A. Levina, J. M. Herrmann, T. G. 2007, *Nature Physics*, 3

[12] Cardy, J. L. & Sugar, R. L. 1980, *J. Phys. A: Math. Gen.*, **13**, L423

[13] Gutenberg, B. & Richter, C. F. 1956, *Ann. Geophys.*, 9

[14] B. D. Malamud, G. Morein, D. L. T. 1998, *Science*, 281

[15] Flyvberg, H. 1996, *Phys. Rev. Lett.*, 76

[16] Gary, C. M. & Singer, W. 1989, *Nature*, 338