Complex intermittency blurred by noise: Theory and application to neural dynamics

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We propose a model for the passage between metastable states of mind dynamics. As changing points we use the rapid transition processes simultaneously detectable in EEG signals related to different cortical areas. Our model consists of a non-Poissonian intermittent process, which signals that the brain is in a condition of complexity, upon which a Poisson process is superimposed. We provide an analytical solution for the waiting-time distribution for the model, which is well obeyed by physiological data. Although the role of the Poisson process remains unexplained, the model is able to reproduce many behaviors reported in literature, although they seem contradictory.

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A low-frequency power spectrum $S(f) \propto 1/f^\eta$ ($0 < \eta < 2$) of a fluctuating signal is a hallmark of complexity as it denotes scale invariance, instead of a characteristic time scale. Its inverse Fourier transform, the autocorrelation function $C(t)$, asymptotically decays as an inverse-power law and its time integral, namely the correlation time, is either null (for $\eta > 1$) or infinite (for $\eta < 1$). These fluctuations are typical of systems at a critical point, where the macroscopic spontaneous dynamics (fluctuations) are long-range correlated and characterized by intermittent behavior [1,2], i.e., driven by *renewal events* [3], namely, fast memory-resetting processes, where entropy production is concentrated. All transport properties, including $S(f)$, can then be written in terms of the waiting-time distribution density $\psi(\tau)$, where τ is the interval between two consecutive renewal events. In particular, for $\psi(\tau)$ with diverging second moment,

$$\lim_{\tau \to \infty} \psi(\tau) \propto \tau^{-\mu} \qquad (1)$$

with $1 < \mu < 3$, it is possible to write simple relations between η and μ [4], thus $S(f)$ is an indirect measure of μ. η can be measured through the use of diffusion: A widely known method, the detrended fluctuation analysis (DFA), is often used to unravel long-range correlations in time series [5]. The search for renewal events is often elusive: most strategies correspond to locate dynamical events within the data (e.g., recurrences or threshold passages) and analyze a symbolic signal stemming from the detected events, eventually comparing, as in [6], the μ index stemming from the analyses with a direct evaluation of the $\psi(\tau)$ of the symbolic sequence. Sometimes the presence of noise forbids a direct comparison with $\psi(\tau)$, and different analyses are used to evaluate μ, as for instance in [7]. Conversely, understanding whether long-range correlations are driven by renewal events is a difficult task, as recently discussed in [8], a paper devoted to distinguish between correlations due to $\psi(\tau)$ (called of Lévy type) and correlations among waiting times.

The brain is a paradigm of complex systems driven by renewal events. The authors of [9] identified rapid transition processes (RTPs) occurring in human electroencephalograms (EEG), namely, almost instantaneous processes gluing otherwise stationary EEG epochs, many of which synchronize different brain areas. Recently Allegrini *et al.* [10] applied both DFA and diffusion entropy (DE) to RTPs of 30 subjects' EEG recording. Subjects were in a relaxed state with closed eyes, instructed to avoid structured thinking. Multichannel (MC) RTPs, i.e., co-occurring in more than one channel, were selected, since they are assumed [9] to mark the beginning or the end of global neural metastable states sustaining neural integration (thus related to consciousness), during which entropy production is minimal. However, the results of [10], i.e., that MC-RTPs are driven by a heavy tailed renewal process with $\mu \approx 2.1$ are, as earlier said, indirect. A knee dividing short- and long-time regimes in the diffusion indicates the presence of uncorrelated noise, thus a direct evaluation of $\psi(t)$ would fail to reveal the μ index.

There have been attempts aimed at measuring the μ of MC RTPs through $\psi(\tau)$. In [11] a direct measure is performed via statistical filtering, and a $\mu \sim 1.6$ is reported. Bianco *et al.* [12] identified metastable-state transitions with topological changes in the minimal-spanning-tree representation of EEG-channels cross-correlations, and found a survival probability

$$\Psi(t) \equiv \int_t^\infty \psi(\tau)d\tau, \qquad (2)$$

following a stretched exponential, $\Psi \sim \exp[-(t/B)^\alpha]$, with $\alpha \sim 0.6$. This result, through the theory of subordination, was shown to correspond to an inverse-power-law renewal process with $\mu = \alpha + 1$, in agreement with [11], but in disagreement with [10]. Both in [11,12], in agreement with [10], evidence is presented for an underlying intermittent, and thus renewal, process.

It is therefore important to establish how the apparent contradiction between [11,12] and [10] can be settled, as far as the values of the index μ is concerned. This will be one of the purposes of the present work, an important task because establishing whether μ is larger than two, as in [10] or smaller, as in [11,12] is crucial, as the value $\mu = 2$ signals,

among renewal models, a transition between two kinds of ergodicity breakdown, stationary and nonstationary, respectively, discussed in [13,14], recently reviewed in [2]. Moreover, complex networks at $\mu \approx 2$ realize the optimized condition for information transfer, due to generalized Linear Response [15]. This condition is obeyed by human language, due to the Zipf's law [6], and by music [12]. A dynamical model of this transition was proposed in [16], proving that $\mu = 2$ optimally allows transmitting and receiving information.

Herein we propose to model MC-RTPs with a superposition of two independent renewal processes: a non-Poisson and a Poisson one. The rationale is that the former describes a global serial process (mentation), that, as in language, produces $\mu \gtrsim 2$ [17], which is in turn blurred by a Poissonian cloud. This cloud, while masking the statistical properties of the sequence, can explain both the Poisson regression at large time-scales, typical of complex systems, and the action of the aged waiting-time distribution. This model, the Copying Mistake Map was originally proposed for modeling DNA sequences [18]. We show that Copying Mistake Map (CMM) is able to reproduce all the aforementioned behaviors concerning $\psi(\tau)$, $\Psi(t)$, and the DFA analysis. We analytically detail the expected results for the various analyses. Then we compare our theoretical CMM predictions with data stemming from Ref. [10].

The CMM model $\{\xi(t)\}$ is the superposition of two sequences, the non-Poisson sequence n, or $\{\xi_n(t)\}$, and the Poisson sequence p, or $\{\xi_p(t)\}$. Both n and p are *renewal*, namely, we first extract the laminar-region sequences $\{\tau^{(n)}(t)\}$, and $\{\tau^{(p)}(t)\}$ as i.i.d. samples from distributions $\psi_n(\tau^{(n)})$, and $\psi_p(\tau^{(p)})$, respectively. We choose $\psi_n(\tau)$ such as $\psi_n(\tau) \propto \tau^{-\mu}$ for $\tau \to \infty$, and $\psi_p(\tau) = r \exp(-r\tau)$. We define the sequences of events

$$t_i^{(n)} = \sum_{k+1}^{i} \tau_i^{(n)} \quad \text{and} \quad t_i^{(p)} = \sum_{k+1}^{i} \tau_i^{(p)}. \tag{3}$$

This means that for each process p or n we have "laminar regions" of mutually independent time duration, with time-instant separations [Eq. (3)]. We construct a signal which is unity in these points, and zero otherwise, namely,

$$\xi_n(t) = \delta_{t,t_i^{(n)}}, \quad \xi_p(t) = \delta_{t,t_i^{(p)}}, \tag{4}$$

$$\xi(t) = \xi_p \vee \xi_n, \tag{5}$$

where $\delta_{i,j}$ is a Kroeneker delta, and $\vee$ is the Boolean OR. As time t is continuous, the probability of having $\xi_p = \xi_n = 1$ is null, and $\xi(t) = \xi_p + \xi_n$. However, in our simulations we use Eq. (5).

We now evaluate $\psi(\tau)$ for the combined sequence [Eq. (5)]. The occurrence of an event can be divided into four complementary situations, depending on the fact that the initial and the final instants of the laminar region can be either a Poisson or a non-Poisson event. We denote with $n(t)$ [$p(t)$] the presence of a non-Poissonian (Poissonian) event at time t. Denoting with $P[a,b]$ the joint probability density for both event a and event b, we write

$$\psi(\tau) = P[n(t),n(t+\tau)] + P[n(t),p(t+\tau)]$$
$$+ P[p(t),n(t+\tau)] + P[p(t),p(t+\tau)]. \tag{6}$$

We now define the probability for an event to belong to the Poissonian, $P(p)$, or to the non-Poissonian series, $P(n)$. We carry out the calculation in the stationary regime, $\mu > 2$. The probabilities $P(p)$ and $P(n)$ are inversely proportional to the respective average times,

$$P(n) \propto \frac{1}{\langle \tau_n \rangle}; \quad P(p) \propto \frac{1}{\langle \tau_p \rangle}, \tag{7}$$

where $\langle \tau_{p,n} \rangle = \int_0^\infty \tau \psi_{p,n}(\tau) d\tau$. After normalization,

$$P(n) = \frac{\langle \tau_p \rangle}{\langle \tau_p \rangle + \langle \tau_n \rangle}; \quad P(p) = \frac{\langle \tau_n \rangle}{\langle \tau_p \rangle + \langle \tau_n \rangle}. \tag{8}$$

The first term in the r.h.s of Eq. (6), $P[n(t),n(t+\tau)]$, is the joint probability of first having a non-Poisson event [$P(n)$], then a non-Poisson event after a time τ and no Poisson events in between, which becomes

$$P[n(t),n(t+\tau)] = P(n)e^{-r\tau}\psi_n(\tau), \tag{9}$$

where the definition $\Psi_p(\tau) \equiv \int_\tau^\infty d\tau' r \exp(-r\tau') = \exp(-r\tau)$ was used. We have used the fact that the Poisson process has a constant rate and, consequently, no aging. Using the same property we can analogously compute $P[n(t),p(t+\tau)]$. We write

$$P[n(t),p(t+\tau)] = P(n)\Psi_n(\tau)re^{-r\tau}. \tag{10}$$

Next, we could use the equality $P[p(t),n(t+\tau)] = P[n(t),p(t+\tau)]$, a consequence of the time-reversal symmetry of both processes and, consequently, also of the global process. We prove this fact by deriving $P[p(t),n(t+\tau)]$ in a direct way, namely by writing

$$P[p(t),n(t+\tau)] = P(p)e^{-r\tau}\psi_n^\infty(\tau), \tag{11}$$

where $\psi_n^\infty(\tau)$ is the infinitely aged waiting-time distribution. In the right-hand side of this expression the first factor denotes the probability of starting from a Poisson event, the second one denotes the fact that no Poisson events occur, while the third term, in line with the other cases, is the probability density of a non-Poisson event occurring after a waiting time τ. Notice that we have lost track of the previous non-Poisson event, and this forces us to use an aged ψ. Due to stationarity assumption, this term is given by the infinitely aged one. In detail [19],

$$\psi_n^{t_a}(\tau) = \int_{-t_a}^0 R(t'+t_a)\psi_n(\tau-t')dt', \quad \text{where}$$

$$R \equiv \sum_{i=0}^\infty \psi_n^i(t), \quad \psi_n^i(t) = \int_0^\infty dt' \psi_n^{i-1}(t-t')\psi_n(t'), \tag{12}$$

$\psi_n^0(t) = \delta(t)$, a Dirac δ. For $t_a = \infty$ $R = 1/\langle \tau_n \rangle$ [19], so

$$\psi_n^\infty(\tau) = \frac{1}{\langle \tau_n \rangle} \int_{-\infty}^0 \psi_n(\tau - t') dt' = \frac{\Psi_n(\tau)}{\langle \tau_n \rangle}. \quad (13)$$

Substituting this relation and the expression for $P(p)$ given in Eq. (8) into Eq. (11), we get

$$P[p(t), n(t + \tau)] = \frac{\langle \tau_n \rangle}{\langle \tau_p \rangle + \langle \tau_n \rangle} \frac{\Psi_n(\tau)}{\langle \tau_n \rangle} e^{-rt}$$

$$= \frac{\langle \tau_p \rangle}{\langle \tau_p \rangle + \langle \tau_n \rangle} \Psi_n(\tau) r e^{-rt} = P(n) \Psi_n(\tau) r e^{-r\tau}, \quad (14)$$

identical to $P[n(t), p(t + \tau)]$, where we used Eq. (8) for $P(n)$ and $r = 1/\langle \tau_p \rangle$. Finally, the probability of having a Poisson event $[P(p)]$, and then a Poisson event after a time τ with no non-Poisson events in between is

$$P[p(t), p(t + \tau)] = P(p) r e^{-r\tau} \Psi_n^\infty(\tau), \quad (15)$$

where, again, the aged Ψ_n^∞ has to be used. Inserting Eqs. (9)–(11), (14), and (15) into Eq. (6) yields

$$\psi(\tau) = \{P(n)[\psi_n(\tau) + 2r\Psi_n(\tau)] + P(p)r\Psi_n^\infty(\tau)\}e^{-r\tau} \quad (16)$$

and the survival probability $\Psi(t) \equiv \int_t^\infty \psi(\tau) d\tau$ can be calculated by direct integration. The result is

$$\Psi(\tau) = P(n)e^{-r\tau}\Psi_n(\tau) + P(p)e^{-r\tau}\Psi_n^\infty(\tau), \quad (17)$$

where $\Psi_n^\infty(t) \equiv \int_t^\infty \psi_n^\infty(\tau)d\tau$.

Notice that before the exponential cutoff in Eq. (16) we have the joint action of two inverse-power laws, with indexes μ and $\mu - 1$. If r were vanishingly small, the asymptotic behavior would be dictated by the index μ. We recall that the theory rests on the case $\mu > 2$. However, when $r > 0$ the presence of an inverse power law with an index smaller than two is numerically evident. Moreover, the presence of two inverse-power decays and the exponential cutoff can mimic a stretched exponential.

Figure 1(a) shows the decay of $\psi(\tau)$ for a typical EEG signal, denoted with a solid-line histogram with logarithmic binning, a choice due to low statistics in the long-time limit. The thick solid line represents Eq. (16) with $\mu = 2.05$, in agreement with [10]. Notably, data are way off the eye guide relative to $\psi(\tau) \propto \tau^{-2.05}$ (dot-dashed line), while a power-law best fit (dotted line) yields an erroneous $\mu = 1.6$ in agreement with [11]. We used

$$\psi(\tau) = (\mu - 1)T^{\mu-1}(T + \tau)^{-\mu}, \quad (18)$$

a form adopted for human cognition in [20], where it was proved to be equivalent to the Fechner's law of logarithmic time perception [21]. Notice that while the asymptotic long-time form of Eq. (16) is well reproduced by data, this is not true in the short-time limit, for $\tau < 30$ ms, due to limitations in the method of extracting events [10].

A discrepancy is even more evident in Fig. 1(b), showing $\Psi(t)$. The solid line, corresponding to Eq. (17) is far from data, due to short-time integration and to low statistics. However we show two sets of data points, one stemming from EEG data (open squares), and another with a simulation of the CMM model (open circles) with the same parameters
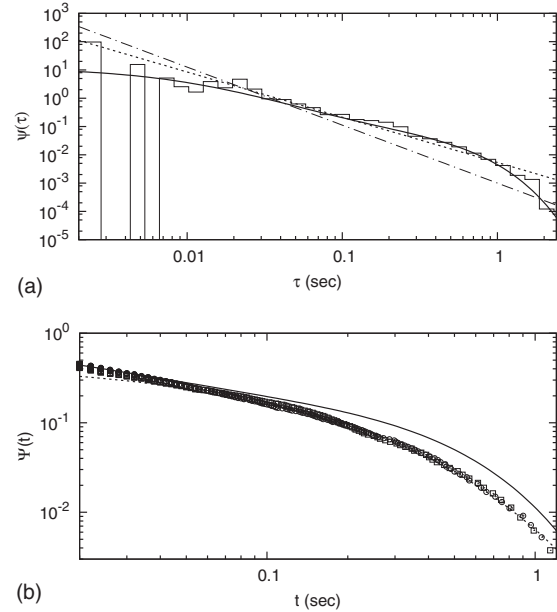

FIG. 1. (a) Waiting-time distribution density. EEG RTPs data are shown with a solid-line histogram. Solid line is theoretical prediction Eq. (16) using Eq. (18) with $\mu = 2.05$, $r = 3.0$ Hz, $T = 6.0$ s. Dot-dashed line is an eye-guide inverse-power law with index 2.05. The dotted line is an inverse-power-law best fit to the data within time interval $[0.02, 2]$s (index 1.6). (b) Survival probability. Solid line is Eq. (17) (same parameters as Fig. 1(a)). Open squares stem from data. Open circles stem from a numerical simulation of the CMM (same parameters as before, same statistics as the data). Dotted line is a stretched exponential $A \exp[-(t/B)^\alpha]$, with $A = 0.5$, $B = 45$ s, $\alpha = 0.6$.

used for Fig. 1(a) and the same length of the data analyzed (5 min, sampling ratio 2 ms). The curves stemming from the two point sets are indistinguishable. Notably, the dotted line, indicating a best stretched-exponential fit for $\Psi(t)$ seem to accurately describe both data sets, as earlier stated. It is in fact indistinguishable from data for $t > 30$ ms, and corresponds to an artifactual $\alpha = 0.6$, in agreement with [12].

Figure 2 shows the outcome of correlational analysis on the CMM model. We build a diffusing trajectory from $\xi(t)$ using two different rules. We make use of DFA to unravel long-range correlations. The correlation function of the signal $\xi(t)$ is proportional to the second derivative of
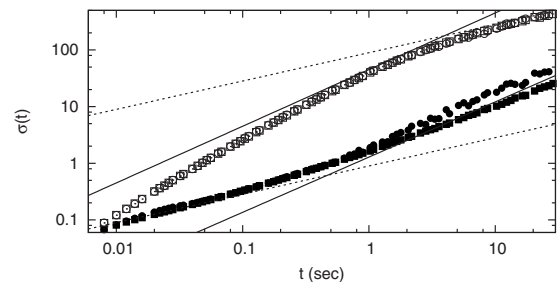

FIG. 2. DFA Analysis: Rule no. 1 for EEG data (●) and simulation (■); Rule no. 2 for EEG data (□) and simulation (□). Solid lines: eyes guide for $\sigma(t) \propto t^{2-\mu/2}$, with $\mu = 2.05$. Dotted lines: eyes guide representing $\sigma(t) \propto \sqrt{t}$.

$\sigma^2(t) \equiv \langle x^2(t) \rangle$, where $\dot{x} = \xi$ or, equivalently $x(t) = \int_{t_0}^{t_0+t} dt' \, \xi(t')$, and average $\langle \cdot \rangle$ is an average over all values of t_0 (moving windows). Thus, if $\langle \xi(t_0)\xi(t_0+t) \rangle$ is not integrable, i.e., it decays as $t^{-\beta}$ with $0 < \beta < 1$, then asymptotically $\sigma^2(t) \sim t^{2H}$, with $H = 1 - \beta/2$.

In Rule no. 1 $\xi(t)$ is simply Eq. (5). As earlier stated, $\xi(t) = \xi_p(t) + \xi_n(t)$ and since the two sequences p and n are mutually independent, it is possible to separate their second moment contributions x_p and x_n and we have that

$$\sigma^2(t) = \langle x_p^2(t) \rangle_p + \langle x_n^2(t) \rangle_n, \tag{19}$$

where the suffix $p(n)$ on the angular brackets means the average on the $p(n)$ sequence alone. The Poisson process yields standard diffusion $\langle x_p^2 \rangle_p \propto t$, while for renewal non-Poisson processes $\langle x_n^2(t) \rangle_n \propto t^{4-\mu}$ [10]. The non-Poisson process dominates in the asymptotic limit. However, the presence of the Poisson process yields a transient $H = 0.5$ in the short-time limit. This behavior is illustrated in Fig. 2, where we compare the analyses stemming from the model and from the same EEG data of Fig. 1.

Rule no. 2 consists of making a walker assume a constant velocity ± 1 within laminar regions, chosen with a coin-tossing procedure for every region. In this case Eq. (19) does not hold: While for this rule we again have $\langle x_p^2 \rangle_p \propto t$ and $\langle x_n^2(t) \rangle_n \propto t^{4-\mu}$ [10], the asymptotics are dominated by the Poisson process, and the non-Poisson scaling is confined at shorter time scales. Figure 2 illustrates this behavior both for the model and the experimental data.

In conclusion, we studied a noisy intermittent model, which can be adopted for many complex systems. The renewal approach provides a formal connection between the consciousness-related events found by [9] and the theory of complex critical phenomena. This approach yields an intermittent entropy production (thus preventing from information explosion) that, in line with the pioneering vision of James [22], allows *thoughts* to be metastable, or temporally discontinuous [9] and with a *limited capacity*, i.e., a significant reduction of degrees of freedom. These renewal properties, hypothesized by [9], are in fact typical of self-organized assemblies. This was proved theoretically [1] and experimentally [2]. The fact that MC-RTPs are driven by renewal processes was provided in [10].

Herein we proved that in fact MC-RTPs are not perfectly renewal, but are in fact the superposition of a Poisson noise and a *single* complex renewal process. The CMM interpretation allows a nonambiguous measurement of the self-organization index μ, and, in doing so, explains the different values reported in literature, erroneously suggesting $\mu < 2$ in resting wakefulness. Notice that the CMM model is crucial to explain both the $\psi(\tau)$ of Fig. 1 and the long-range correlations of Fig. 2. A blind renewal modeling only based on the power spectral analysis would yield the dot-dashed line of Fig. 1(a).

The presence of a single non-Poissonian renewal process in unconstrained EEG activity may provide the serial mental processing hypothesized in [23] to integrate the unconscious parallel processing occurring in different brain areas. It is worth noticing that such process is a product of self-organization, rather than by the hierarchical process. Since (results will be reported elsewhere) single-channel transitions, although with similar $\psi(\tau)$, do not obey the simple CMM dynamics [Eq. (5)], we claim that a serial binding activity among neural groups (intermittent integrated phase-lock dynamics) stands at the basis of the holistic output called consciousness.

[1] Y. F. Contoyiannis and F. K. Diakonos, Phys. Lett. A **268**, 286 (2000).
[2] L. Silvestri, L. Fronzoni, P. Grigolini, and P. Allegrini, Phys. Rev. Lett. **102**, 014502 (2009).
[3] D. R. Cox, *Renewal Theory* (Methuen & Co. Ltd., London, 1962).
[4] S. B. Lowen and M. C. Teich, Phys. Rev. E **47**, 992 (1993).
[5] C.-K. Peng *et al.*, Phys. Rev. E **49**, 1685 (1994).
[6] P. Allegrini, P. Grigolini, and L. Palatella, Chaos, Solitons Fractals **20**, 95 (2004).
[7] N. Scafetta and B. J. West, Phys. Rev. Lett. **92**, 138501 (2004).
[8] D. Rybski *et al.*, Proc. Natl. Acad. Sci. U.S.A. **106**, 12640 (2009).
[9] A. A. Fingelkurts and A. A. Fingelkurts, Brain Mind **2**, 261 (2001); Lang. Cognit. Processes **7**, 135 (2006).
[10] P. Allegrini *et al.*, Phys. Rev. E **80**, 061914 (2009).
[11] P. Gong, A. R. Nikolaev, and C. van Leeuwen, Phys. Rev. E **76**, 011904 (2007).
[12] S. Bianco *et al.*, Phys. Rev. E **75**, 061911 (2007).
[13] M. H. Lee, Phys. Rev. Lett. **98**, 110403 (2007); Acta Phys.

Pol. B **39**, 1035 (2008).
[14] G. Margolin and E. Barkai, J. Stat. Phys. **122**, 137 (2006).
[15] B. J. West, E. L. Geneston, and P. Grigolini, Phys. Rep. **468**, 1 (2008).
[16] R. F. Cancho and R. V. Solé, Proc. Natl. Acad. Sci. U.S.A. **100**, 788 (2003).
[17] J. M. Medina, Phys. Rev. E **79**, 011902 (2009).
[18] P. Allegrini, P. Grigolini, and B. J. West, Phys. Lett. A **211**, 217 (1996).
[19] P. Allegrini, G. Aquino, P. Grigolini, L. Palatella, and A. Rosa, Phys. Rev. E **68**, 056123 (2003).
[20] T. Takahashi, H. Oono, and M. H. B. Radford, Physica A **387**, 2066 (2008).
[21] G. T. Fechner, *Elemente der Psychophysik* (Breitkopf und Hartel, Leipzig, 1860).
[22] W. James, *The Principles of Psychology* (Dover, New York, 1890), Vol. 1.
[23] B. J. Baars, in *CIBA Symposium on Experimental and Theoretical Studies of Consciousness*, edited by G. R. Bock and J. Marsh (Wiley, London, 1993), p. 282.