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A chaotic model of migraine headache considering the dynamical transitions of this cyclic disease

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Abstract – Migraine is one of the primary headache disorders in a group of the ten most prevalent and disabling diseases. There are some valuable computational models of this disease which considered the onset and spatial patterns of migraine pain. Here we focus on dynamical transitions of this cyclic disease using the subnetworks which are essential in its complex network. Regarding the dynamical diseases theory, we propose a dynamical network biomarker for this disease that can predict the upcoming prodromal phase for clinical use. In this research, we use the bifurcation diagram as a tool to show the prediction of the model as the considered physiological parameter of the model changes.

Introduction. – Migraine headache is the third most prevalent and sixth most disabling disease in the world. This cyclic disease is characterized by repetitive periods of throbbing and severe pain in one side of the head. Each migraine cycle can be separated into one normal phase (inter-ictal) and four distinct abnormal phases; prodromal, aura, ictal and postdrome phases. Each phase has its own associated symptoms, prevalence, and duration which are different between migraineurs. Considering the existence of each phase in the migraine cycle, there are several subtypes of this disease such as migraine with aura (MA) and migraine without aura (MWOA), which are major subtypes of migraine headaches.

Despite the research that has been done about this disease, the mechanism of migraine is not yet well understood. There are two main theories about the migraine mechanism [1]: Spreading Depression (SD) and Migraine Generator (MG) theories. The SD theory of migraine assumes that Cortical Spreading Depression (CSD) waves cause neurological symptoms and pain in the aura and ictal phases, respectively [2,3]. However in MG theory, a large amount of evidence supports the claim that dysfunction in the brainstem causes the migraine cycle and even CSD waves [4,5]. Many different studies have shown that neural hyperexcitability is the physiological parameter that causes the migraine cycle to initiate. Actually, increasing excitability brings the brain to a tipping point in which a migraine initiates [6–8].

Mathematically, migraine is a dynamical disease containing transitions that happen because of parameter changes through the migraine cycle [1,6,9]. To investigate the progress of this disease, computational models can be used. These models should characterize the essential involved subnetworks and their connections during the migraine cycle which are needed to understand how the migraineur's brain can reach a tipping point through increasing excitability.

Dynamical diseases, the extension of periodic diseases, are defined as those in which changes in physiological control parameters cause sudden transitions and abnormal dynamics [10]. Transitions between phases in the migraine cycle put this disease in a group of dynamical diseases. The concept of bifurcation can mathematically describe a dynamical disease with critical transitions [6,10–15]. In complex diseases with distinct phases, the Dynamical Network Biomarker (DNB) is essentially a group of states that are highly deviated from each other. The biological systems show DNB at critical transition points [15–20]. A DNB which is strongly correlated and has high-amplitude fluctuating states has been seen in the pre-ictal phase of dynamical diseases and migraine which we focus on in this paper and has not been seen before. Moreover, regarding

the complex dynamical diseases concept, both inter-ictal and ictal phases are steady states with low-amplitude fluctuation and correlation in states [1,16].

Accurate prediction of transitions will improve the quality of treatment strategies in patients with dynamical diseases. A lot of effort has been made to propose indicators which can detect the vicinity of critical transitions in ecological, financial and biological phenomena [21]. Among the proposed indicators, variance, skewness and autocorrelation at-lag-1are most commonly employed to predict the critical transitions in experimental and simulated data [15].

Habituation which still is not completely understood, is a physiological learning process in which there is a decrease in amplitude of response to a repeated stimulus. Habituation changes during the migraine cycle is one of the most reproducible experiments in migraines. Neurophysiological studies have shown that in most methodological approaches, the migraineur's brain shows pre-ictal and postdrome abnormality in habituation. Interestingly, just before and during the ictal phase, habituation normalizes like inter-ictal phase [22]. These results, in addition to complex dynamical disease concepts, can be used to investigate dynamical features of inter-ictal and ictal phases.

Chaos occurs in complex systems whose behavior is highly sensitive to initial conditions. Consequently, small changes in initial conditions can yield completely different responses [23]. Some evidence claims that chaos exists in many biological systems both in normal and abnormal situations, *e.g.* brain [24–31], heart [32,33], and kidney [34]. According to this evidence, chaotic behavior is considered in many computational models of different biological systems [11,12,35–41]. Recently dynamical systems were categorized into systems with self-excited attractors and systems with hidden attractors [42–46]. When an attractor's basin of attraction involves equilibrium, we call that attractor "self-excited". Otherwise, the attractor is hidden [47–50]. Through the nonlinear dynamics concepts such as chaos, bifurcation diagram, hidden attractors and tipping points, we try to make effort to better understand the mechanism of migraine headache.

Migraine model. –

Dynamical and structural assumptions. As mentioned in the previous section, the migraine (MWOA type) cycle is considered as four distinct phases each having its own dynamic (fig. 1). In the attack-free interval (normal phase), as the brain locates in its normal states, chaotic dynamics are considered. This dynamic not only provides low-amplitude fluctuation and low-correlation in states but also confirms by experiments that the brain is chaotic in normal states. It should be noted that the deviations of the states are low and the neighboring states are just close to each other as we see this type of responses in chaotic attractors. However, in the pre-ictal phase, these fluctuations are more deviated and the states are far from each other as we see this type of responses in periodic attractors; hence, periodic dynamics can provide strongly correlated and high-amplitude fluctuating states in DNB which are seen in the pre-ictal phase. As mentioned before, the ictal phase has the same chaotic dynamics as the inter-ictal one. In the postdrome phase, a physiological parameter goes through the opposite direction that it passed during the three past phases. But in this case, the brain does not repeat the exact previous responses. In nonlinear dynamics theory, there are coexisting attractors in this situation, and hysteresis happens between these attractors.

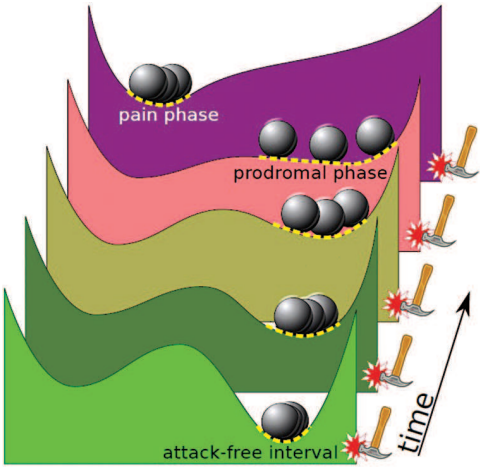

Fig. 1: (Colour online) Migraine cycle illustration. In this time-variant landscape, the balls represent the current state and its variability (yellow dashed line) as the excitability of the brain increases over time. Also stable attractors, both pain state and attack-free states, are represented with potential walls. At the end of the pain state, when the trigger factors (hammer) end, the curvatures of the two wells change in the way that the balls leave the left well and go to the right well, and subsequently the postdrome phase occurs; hence, to consider the postdrome phase, we should consider the same landscapes, but at this time, from top (pain phase) to bottom (attack-free interval). The second and third landscapes show the transient states between attack-free interval and prodromal phase. Regarding the amplitude of the fluctuations (fluctuations of the balls), these two states can be categorized to attack-free interval or prodromal phase. Modified figure from fig. 1 of ref. [9].

Previous studies suggested the role of different parts in the migraine generator network [4,5,51,52]. Despite the value of considering all details of this network, it is better to consider the simplest network with essential parts in which transitions occur as control parameters change [53]. MGN can be considered as a system containing two subnetworks; the Trigeminovascular and the descending modulatory brainstem systems [53]. The link between the MGN and SD theories are probably irreconcilable since these two subnetworks should be connected to the cortex where SD happens (fig. 2).

Local population of neurons model for migraine. Our mathematical model is based on [6] which will be briefly explained in this subsection. In this model, activity (A) of

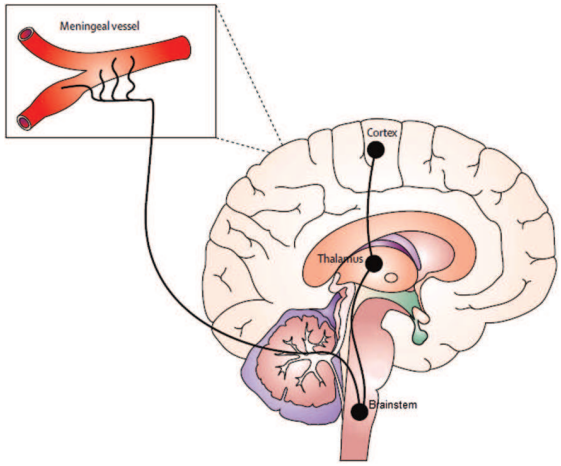

Fig. 2: (Colour online) Migraine network. Trigeminovascular, descending modulatory brainstem and cortex are considered as the main subnetworks of this model. Modified figure from fig. 2 of ref. [54].

a local population of neurons is considered to be the result of interactions between the generation and decay of pulses. The following equation describes the neural activity rate:

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \left(\varepsilon S + A \cdot q \frac{\varepsilon^p}{\varepsilon^p + \varepsilon_{crit}^p}\right)(1 - A) - d \cdot A \qquad (1)$$

In this equation, the last term $(d \cdot A)$ reflects the decay of pulses with a rate d, and the rest of this equation represents the generation of pulses. In the generation term, $1 - A$ is zero when A equals one so that activity belongs to the interval $(0, 1)$. The multiplier $(\varepsilon S + A \cdot q \frac{\varepsilon^p}{\varepsilon^p + \varepsilon_{crit}^p})$ illustrates two main terms that cause generation of pulses; the first one, external stimuli (εS) in which excitability of the neurons (ε) increases this effect, and the second one is the activity of the neighboring neurons $(A \cdot q \frac{\varepsilon^p}{\varepsilon^p + \varepsilon_{crit}^p})$. In the neighboring term, the factor q is the maximum intensity of its effect, and the Hill function $(\frac{\varepsilon^p}{\varepsilon^p + \varepsilon_{crit}^p})$ is used to show that this effect dominates when ε reaches a critical excitability level (ε_{crit}). The parameter p is the Hill function coefficient. For simplicity, the activity of the neighboring region (A in neighboring term) is considered equal to the activity of the main local region.

Also one of the main assumptions in the model is the feedback effect of activity on excitability, which is formulated with a linear equation as follows:

$$\varepsilon = \varepsilon_0 + cA, \qquad (2)$$

where ε_0 is the base-line excitability and c is a constant. The full model is defined by eq. (3) which is constructed by substituting eq. (2) into eq. (1):

$$\frac{\mathrm{d}A}{\mathrm{d}t} = ((\varepsilon_0 + cA)S + A \cdot q \frac{(\varepsilon_0 + cA)^p}{(\varepsilon_0 + cA)^p + \varepsilon_{crit}^p})(1 - A) - d \cdot A \qquad (3)$$

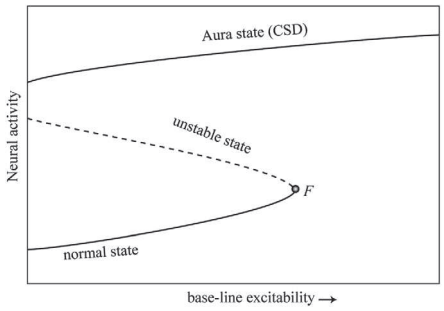

Fig. 3: (Colour online) Representation of how stable and unstable equilibrium values change as a function of base-line excitability. The fold bifurcation point (F) determines the base-line excitability value at which the normal state loses its stability and the disease state dominates. Modified figure from fig. 2(c) of ref. [6].

This model is based on considering ignition of spreading depression as the pathophysiological mechanism for migraine initiation. Increasing excitability brings the brain to a tipping point which causes SD to start. According to [6], up to three stable and unstable equilibria are generated from eq. (3) as the parameter ε_0 changes. Figure 3 shows values of these equilibria and stability of each one as a function of ε_0. In fig. 3, the dashed middle branch characterizes the unstable equilibrium which determines the border between normal and disease stable equilibria. As base-line excitability of the brain increases to a critical level, the unstable equilibrium collides with the normal stable equilibrium and causes the disease equilibrium to dominate. In fig. 3, F is the critical level at which a fold-bifurcation occurs and the brain enters the disease state from a pathophysiological point of view.

The proposed model. To consider the migraine generator as a network with complex interactions, three units, specifying the Trigeminovascular, descending modulatory brainstem and cortex, are coupled. In this model, each population of neighboring neurons is considered as a distinct unit. A linear summation with constant coefficients of all activities is used to illustrate neighboring interactions. This coupling method is illustrated for i-th unit as follows:

$$A_{ni} = K_{i1}A_1 + K_{i2}A_2 + K_{i3}A_3 \qquad i = 1, 2, 3. \qquad (4)$$

A_{ni} is the activity of a population of neighboring neurons for the i-th unit. Also K_{ij} is the multiplier of j-th unit in i-th one. The complete model is

$$\begin{aligned}
\frac{\mathrm{d}A_1}{\mathrm{d}t} &= \Big((\varepsilon_{01} + c_1A_1)S_1 + (K_{11}A_1 + K_{12}A_2 + K_{13}A_3) \\
&\quad \cdot q_1 \cdot \frac{(\varepsilon_{01} + c_1A_1)^{p_1}}{(\varepsilon_{01} + c_1A_1)^{p_1} + \varepsilon_{crit1}^{p_1}}\Big)(1 - A_1) - d_1 \cdot A_1, \\
\frac{\mathrm{d}A_2}{\mathrm{d}t} &= \Big((\varepsilon_{02} + c_2A_2)S_2 + (K_{21}A_1 + K_{22}A_2 + K_{23}A_3) \\
&\quad \cdot q_2 \cdot \frac{(\varepsilon_{02} + c_2A_2)^{p_2}}{(\varepsilon_{02} + c_2A_2)^{p_2} + \varepsilon_{crit2}^{p_2}}\Big)(1 - A_2) - d_2 \cdot A_2,
\end{aligned}$$

$$\frac{\mathrm{d}A_3}{\mathrm{d}t} = \Bigg((\varepsilon_{03} + c_3A_3)S_3 + (K_{31}A_1 + K_{32}A_2 + K_{33}A_3) \cdot q_3 \cdot \frac{(\varepsilon_{03} + c_3A_3)^{p_3}}{(\varepsilon_{03} + c_3A_3)^{p_3} + \varepsilon_{crit3}^{p_3}}\Bigg)(1 - A_3) - d_3 \cdot A_3. \tag{5}$$

Parameters S_i, p_i, q_i and d_i are set equal to the values in [6] ($S_i = 0.1$, $p_i = 4$, $q_i = 1$ and $d_i = 0.1$). Other parameters are set such that the model becomes simpler. It should be noted that other choices for values of these parameters are also available, but the goal of this work is not investigating the quantitative values of these parameters, and it is sufficient that this physiologically meaningful model shows the migraine cycle phases and their dynamics.

Numerical results and discussion. – The proposed model is simple with a few parameters which are essential to show the dynamical transitions in the migraine cycle. According to the physiological assumptions section, these three subnetworks should show the considered dynamics in each phase of the migraine cycle. As mentioned above, basal excitability of the first subnetworks (ε_{01}) of this model is considered as a bifurcation parameter when the other parameters are fixed at values chosen by trial and error as $\varepsilon_{02} = 1$, $\varepsilon_{03} = 1$; $c_1 = 1$, $c_2 = 1$, $c_3 = 1$; $K_{11} = 0$, $K_{12} = -1$, $K_{13} = -7$, $K_{21} = 1$, $K_{22} = 0$, $K_{23} = 0$, $K_{31} = 23$, $K_{32} = 0$, $K_{33} = 0$; $\varepsilon_{crit1} = 1$, $\varepsilon_{crit2} = 1$, $\varepsilon_{crit3} = 1$. To evaluate the transitions in one cycle of this disease, a bifurcation diagram of the model is shown in fig. 4.

The left chaotic region of the fig. 4 ($1.39 < \varepsilon_{01} < 1.403$) represents the attack-free interval. The periodic region shows the prodromal phase ($1.403 < \varepsilon_{01} < 1.428$). Also the second chaotic region shows the pain phase ($1.428 < \varepsilon_{01} < 1.45$). The black line which represents the postdrome phase ($1.45 > \varepsilon_{01} > 1.39$) of the migraine is the coexisting attractor which emerges as the bifurcation parameter decreases gradually. The prodromal and postdrome phases show period-3 and period-1 responses, respectively. It should be noted that the model does not show the aura phase; hence the model represents MWOA dynamics.

In such a dynamical disease, an indicator is needed to show the migraine progress from the inter-ictal state to the ictal state through the prodromal state. To identify each phase, the variance of local maxima which was considered as a "migraine phase indicator" is calculated. The transitions get the state of the system away from its stable state and make a large drift which increases the variance of the system. Particularly, it shows the deviation of the brain state through smooth changes of excitability.

Recently dynamical systems were categorized into systems with self-excited attractors and systems with hidden attractors. When an attractor's basin of attraction involves equilibrium, we call that attractor "self-excited". Otherwise, the attractor is hidden. Rather than design, their localization and control have been of great interest in recent years [55–57]. Although hidden attractors exist

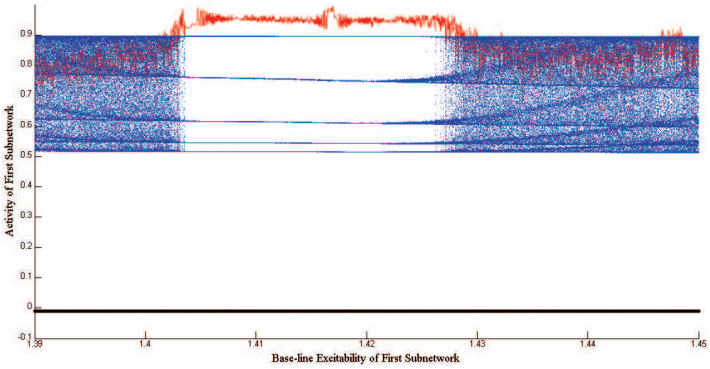

Fig. 4: (Colour online) The model bifurcation as the base-line excitability (ε_{01}) of the first subnetwork increases gradually. The first chaotic region of the figure represents the attack-free interval, and the periodic region shows the prodromal phase. Also the second chaotic region shows the pain phase. The black line which represents the postdrome phase of the migraine is the coexisting attractor which emerges as the bifurcation parameter decreases gradually. It should be noted that the model does not show the aura phase; hence the model represents MWOA dynamics. Variance of the local maxima (in red) is the dynamical biomarker which predicts the upcoming prodromal phase.

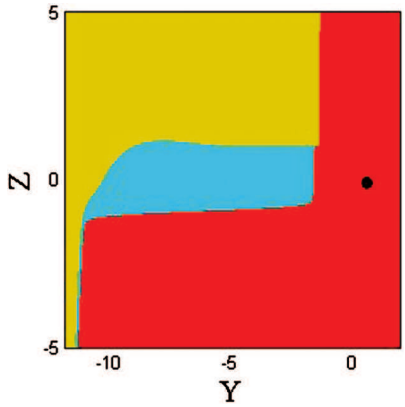

Fig. 5: (Colour online) Cross section of the basins of attraction of the two attractors (chaotic attractor and fixed-point attractor) in the yz-plane at $x = 0$ for the proposed system. The blue area is the basin of the chaotic attractor, the red area is the basin of the fixed-point attractor (shown with a black circle), and unbounded regions are shown in yellow.

in some rare real-world dynamical systems [58,59] no such system has been reported in biological system. Here we show that the chaotic strange attractor in the proposed system is hidden. One easy way to check that is to plot the basin of attraction around the equilibrium point (which is obtained numerically and has been plotted as a black circle in fig. 5). It can be observed from fig. 5 that the basin of attraction for the strange attractor does not intersect with the equilibrium point. Thus, according to definition, the strange attractor is hidden.

Conclusion. – In this study, a novel behavioral model of migraine is proposed which involves an essential

subnetwork of the complex network of a migraine. This model has the potential to present each phase with its specific considered dynamic which resembles experimental studies and the theory of dynamical diseases. In this type of model, the bifurcation diagram is the tool to show the dynamic changes of the model as the key parameter of the model increases and/or decreases.

Defining dynamical network biomarkers is done in some dynamic diseases, but we have not seen application of these biomarkers in migraines. In this study we introduce variance of local maxima as the DNB to predict the emergence of the upcoming phase of the migraine. This DNB increases just before the prodromal phase and can be used as an early warning signal in this disease.

Other improvements to the model would involve consideration of time delays which exist in the coupling of subsystems [60–65] and synchronization methods in this chaotic network [61,64–67]. Larger network sizes [40,68,69] and electromagnetic field effect [39,40,70,71] are two lines of research which should be considered in future works.

* * *

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