Do strokes affect the brain's critical state? A theoretical perspective.

Jakub Janarek[1,2], Zbigniew Drogosz[1,2], **Jacek Grela**[1,2], Jeremi K. Ochab[1,2], Pawel Oswiecimka[1,2,3], Maciej A. Nowak[1,2], Dante R. Chialvo[4,5]

[1] *Institute of Theoretical Physics, Jagiellonian University, 30-348 Kraków, Poland*

[2] *Mark Kac Center for Complex Systems Research, Jagiellonian University, 30-348 Kraków, Poland*

[3] *Complex Systems Theory Department, Institute of Nuclear Physics, Polish Academy of Sciences, 31-342 Kraków, Poland*

[4] *Center for Complex Systems & Brain Sciences (CEMSC^3), Escuela de Ciencia y Tecnología, Universidad Nacional de San Martín, Buenos Aires, Argentina*

[5] *Consejo Nacional de Investigaciones Científicas y Tecnológicas (CONICET), Buenos Aires, Argentina*

Abstract. In recent tests of brain criticality in stroke patients, it was suggested that lesions cause a non-critical state of neural dynamics, and the critical state might subsequently be restored in parallel with a patient's post-stroke behavioral recovery (Rocha et al.). We propose an alternative interpretation in which the brain remains in the critical state at all times; however, as a result of a stroke, it may effectively become divided into two or more weakly connected regions, mimicking the lack of criticality. This interpretation is corroborated by toy simulations of the Ising model and a more realistic Haimovici-Tagliazucchi-Chialvo model based on the Hagmann et al.'s connectome with "artificial strokes" performed by removing connections between two subsystems. In such models, standard indicators of criticality based on cluster size analysis are found to behave similarly to those in models based on real-world MRI scans of stroke patients. Our study suggests that the lack of the peak in the second largest cluster, signaling the loss of criticality, may be an artifact of the division of the original system into weakly connected parts.

Introduction

A canonical example of a complex system is the human brain, whose large numbers of neuronal cells display nontrivial multiscale organization and complex characteristics. The concept of the critical brain suggests that neural networks evolve towards a critical state, where the competition between order and disorder states emerges. Such a system behavior indicates optimization of computational properties related to information processing, such as information transmission and storage or computational power, which is especially appealing in neurosciences [1].

The ideas of critical phenomena have recently been applied to the study of the brain dynamics of stroke patients by Rocha et al. [2]. Their most intriguing result was the fact that the presence and severity of the stroke were related to loss of critical behavior in the brain and possible post-stroke recovery of a patient to the recovery of criticality. Their results were based on the analysis of the sizes of largest clusters of activity. We revisit these findings from a theoretical perspective by studying artificial "strokes" in the toy Ising model and the HTC model.

Results and discussion

In this work, we demonstrate a simple mechanism whereby a critical system consisting of at least two weakly connected parts may appear non-critical to the usual structure-agnostic analysis of the size of the second largest cluster of activity. We replicate this behavior in a set of numerical experiments on real connectomes with a realistic model of brain activity, where artificial "strokes" are introduced by removing some connections between regions of the standard healthy connectome. Furthermore, a finer cluster analysis applied to substructures induced by such strokes reveals the criticality hidden from the structure-agnostic analysis. The results of these experiments bear a close resemblance to the results of simulations based on real-world data from stroke patients. We therefore conclude that by itself the second largest cluster size is not a reliable indicator of criticality when applied to a system with unknown subsystem structure. We further argue that stroke-induced loss of criticality may be only illusory and that the described mechanism is a possible explanation for the previous findings. In light of our work, complementary indicators, such as the autocorrelation coefficient, the eigenvalues of the correlation matrix, or the modularity analysis of the connectome, are needed to make criticality analysis more robust.

References

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