

Fight and flight: how long will we lose this game of chess? Pain and autonomic nervous system

Luta e fuga: quanto tempo vamos perder este jogo de xadrez? Dor e sistema nervoso autônomo

DOI 10.5935/2595-0118.20220042-en

Pain is an increasingly studied topic around the world. Not only about defining what pain is, but also the factors that influence its occurrence. Is feeling pain something natural? Or is it just “normal”? An ordinary thing! Well, it depends on which way pain clinician or student directs their gaze. With that, we can say that the nervous system is “the key” of pain management. After all, how to feel pain without the nervous system? How is it possible to feel any sensation, whether of pleasure or not, without it? And, thinking on this way, it is known that both central nervous system (CNS) and somatic nervous system (SNS) are responsible for interpreting, recognizing and modulating potential pain stimuli^{1,2}. Sounds like simple, right? but it's not!!! Then, we would like to invite you to think of pain management as a game of chess. So, we wonder if there is something missing from this game! Well, we strongly believe so; and this “something” is the autonomic nervous system (ANS).

Many people subjugate the intimate “pain and ANS” relationship and we understand why that happens. ANS is commonly interpreted as “viscera’s nervous system”, especially when we refer to heart and intestines. So, it is considered a primitive nervous system. And this “primitive” speech brings a negative terminology about this system, by the way, extremely complex. After all, the CNS is supposed to be specialized; it will never be considered primitive. And, at this point, we draw your attention to the expressive number of receptors, physiological and hormonal pathways and autonomic nerve endings present in the CNS. And we're not just talking about the brainstem, the bulbar topography, but the telencephalon. The brain!

We emphasize here that it is not the fact that autonomic structures are present at the supraspinal level that supports the argument that the ANS is linked to pain. But there is interaction between these systems that interfere in the interpretation of pain, as a sensation and even an emotion. And this fact is closely linked to central sensitization³. Autonomic neurons present in the hippocampus lead to the maintenance of pain memory. Limbic cortex and amygdala are strongly responsible for pain sensation, and these are areas mostly modulated by autonomic pathways⁴. Let's go deeper; N-methyl-D-aspartate (NMDA), glutamatergic receptors, are hyper-excited, for example, in situations that demand our surveillance, and they are what keep us in the focus of attention. “Fight and flight”, two typical behaviors of the ANS function description are only allowed because we have high glutamatergic discharge.

In this discreet reasoning, we hope to have managed to draw your attention to the point of view in which central pain sensitization is modulated by the ANS. We elevate the discussion beyond the doors of acute pain. We are talking about pain chronification and we recommend that you relate what has been said with the concept of nociplastic pain⁵. The correlation is clear, isn't it? After all, how can pain modulation system go wrong, causing your nerve circuitry to derange in a chronic but non-specific way? In other words, for no real reason? Or grounded? Is it because we are still “stubborn” in turning our eyes to the CNS and the sympathetic SNS only? Is it because we can see nerves, spinal cord, brain mass, receptors, but not *neurotags*? Neural networks.

It is something to think about, or rather, it is something to reason about, research, investigate and soon bring to clinical practice. Finally, we invite you, whether a researcher, clinician, pain scholar, whatever, to sit down once again at the front of this chessboard, where we try to understand the pieces to be moved and to treat our patient's pain and consider the SNA pieces in the play. Perhaps, the game will become more complex due to the addition of this whole new system, however, the chances of winning the play will grow strongly.

Josimari Melo DeSantana

Federal University of Sergipe, Department of Physical Therapy, Graduate Program in Health Sciences, Graduate Program in Physiological Sciences. Aracaju, SE, Brazil.

<http://orcid.org/0000-0003-1432-0737>

E-mail: editor.brjp2223@dor.org.br

Annanda Oliveira Santos

Federal University of Sergipe. Graduate Program of Physiological Science. Aracaju, SE, Brazil.

<http://orcid.org/0000-0001-6100-9621>

E-mail: annandaoliveiracel@gmail.com

REFERENCES

1. Mendell LM. The path to discovery of windup and central sensitization. *Front Pain Res.* 2022;3:833104.
2. Crofford LJ. Chronic pain: where the body meets the brain. *Trans Am Clin Climatol Assoc.* 2015;126:167-83.
3. Yeater TD, Clark DJ, Hoyos L, Valdes-Hernandez PA, Peraza JA, Allen KD, et al. Chronic Pain is associated with reduced sympathetic nervous system reactivity during simple and complex walking tasks: potential cerebral mechanisms. *Chronic Stress* Thousand Oaks Calif. 2021;2021;5:24705470211030270.
4. Hohenschurz-Schmidt DJ, Calcagnini G, Dipasquale O, Jackson JB, Medina S, O'Daly O, et al. Linking pain sensation to the autonomic nervous system: the role of the anterior cingulate and periaqueductal gray resting-state networks. *Front Neurosci.* 2020;2020;14:147.
5. Fitzcharles MA, Cohen SP, Clauw DJ, Littlejohn G, Usui C, Häuser W. Nociceptive pain: towards an understanding of prevalent pain conditions. *Lancet.* 2021;397(10289):2098-110.

