



Hyperresponsivity in migraine: a network dysfunction or an analytic cognitive style-connected feature?

Marzia Buonfiglio¹ · Giuliano Avanzini² · Filippo Brighina³ · Francesco Di Sabato¹

Received: 11 August 2018 / Accepted: 27 September 2018 / Published online: 2 October 2018
© Springer-Verlag Italia S.r.l., part of Springer Nature 2018

Dear Editor,

We read the interesting article by Ambrosini, recently published in *Neurological Sciences* [1], summarizing the main recognized mechanism underlying pathophysiology of migraine.

As the author stressed, every effort in the last decades, aimed to unmask any consistent and permanent structural disturbance of the brain of migraine sufferers, was in vain, except for an interictal CNS dysfunction that normalizes ictally, identifiable as a lack of habituation to cortical repeated stimulations [1]. Lack of habituation, even if recently the subject of some criticism [2], has been indeed consistently reported and considered as a main feature of the migrainous brain, at the extent to be proposed as a diagnostic tool so far [1].

However, during the last years, we first provided evidence [3, 4] highlighting a specific, i.e., analytic, style of processing visual and auditory information in a large sample of migraineurs [3]; then, we showed a clear link between this cognitive behavior factor—per se—and lack of habituation through visually evoked potentials [4]. It should be noted that analytic style of processing visual stimuli has shown—per se—the following cortical responsiveness features, similarly to migraine [4]:

- Reduced N1-P1 first block amplitude
- Potentiation instead of habituation to the successive block of responses to visual inputs delivered [4].

✉ Marzia Buonfiglio
marziabuonfiglio@libero.it

¹ Department of Clinical Medicine, Policlinico Umberto I Hospital, Sapienza University of Rome, Rome, Italy

² Department of Neurophysiology and Experimental Epileptology, Carlo Besta Neurological Institute, Milan, Italy

³ Department of Experimental Biomedicine and Clinical Neuroscience (BioNeC), University of Palermo, Palermo, Italy

Whatever the basis of such dysfunction could be, whether depending upon a thalamocortical-dysrhythmia or following to neuronal hyperexcitability [1], it should be noted that a cognitive behavior, i.e., analytic style, strongly associated to migraine [3], is—per se—also characterized by electrophysiologic pattern, i.e., hyperresponsivity, very similar to that found in migraine [4].

In the light of the above considerations, we believe that, when including healthy controls in neurophysiological study on migraine, we cannot disregard and should control for such a cognitive behavioral variable, i.e., analytic information processing style [3], to avoid potential biases in the interpretation of the results.

Moreover, it should be noted that recent report by Lisicki [5], firstly investigating visually evoked responses of migraine patients using single trial analysis, showed anatomofunctional correlates of visual hyperresponsivity (in terms of gray matter volume), especially in the ventral visual attention network. This cerebral network, indeed, has been previously indicated as specialized for analytic (local) visual processing, whereas synthetic (global) aspects of visual information are considered to pertain to dorsal visual network activity [6].

In conclusion, we believe that the above reported cognitive behavioral data, linked to neurophysiological evidence [1, 3–6], justify further research on this issue. As cognitive information processing style can be effectively modulated by appropriate cognitive behavioral training [4], a potential new clinical therapeutic target can also be speculated.

Therefore, both migraine research and therapy might advantage from a multidisciplinary perspective electing the link between this cognitive behavior factor, i.e., analytic style, migraine disease, and the shared neurophysiologic patterns, as an important piece of a yet unsolved puzzle, investigating the meaning of this association from a cognitive behavioral, neurophysiological, and epigenetic view.

This may help us to shed some more light on migraine etiology that stands as a persisting enigma over the centuries.

Acknowledgements The authors would like to thank Giovanna Castelli and Luca Pierotti for their technical assistance.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest.

References

1. Ambrosini A (2018) Neurophysiology of migraine. *Neurol Sci* 39(Suppl 1):559–560
2. Brighina F, Cosentino G, Fierro B (2016) Habituation or lack of habituation: what is really lacking in migraine? *Clin Neurophysiol* 127(1):19–20
3. Di Sabato F, Buonfiglio M, Mandillo S (2013) Analytic information processing style in migraineurs. *Neurol Sci* 34:1145–1150
4. Buonfiglio M, Toscano M, Puledda F, Avanzini G, Di Clemente L, Di Sabato F, Di Piero V (2015) Lack of habituation of evoked visual potentials in analytic information processing style: evidence in healthy subjects. *Neurol Sci* 36:391–395
5. Lisicki M, D’Ostillo K, Coppola G, De Noordhout AM, Parisi V, Schoenen J, Magis D (2018) Brain correlates of single trial visual evoked potentials in migraine: more than meets the eye. *Front Neurol* 9:393. <https://doi.org/10.3389/fneur.2018.00393>
6. Leaver EE, Low KA, DiVacri A, Merla A, Fabiani M, Gratton G (2015) The devil is in the detail: brain dynamics in preparation for a global-local task. *J Cogn Neurosc* 27(8):1513–1527. https://doi.org/10.1162/jocn_a_00800