Response to Letter Regarding Article
“Jugular Veins in Transient Global Amnesia: Innocent Bystanders”

Response:

We read with interest the letter by Griebe et al entitled “Stressing the Pathophysiology of Transient Global Amnesia”1 in which the authors suggest that transient global amnesia (TGA) might be a disorder caused by a stress-related transient inhibition of memory formation in the hippocampus. For this reason they are currently studying the effect of experimental exposure to stress on cortisol levels and on hippocampal activation patterns using functional MRI in patients with TGA.

Stress is often reported as the cause of many psychological and physical problems. All of us have experienced stress at one point or another in our lives and are aware that stress might have important effects on our memory. However, it is not easy to define stress and it is even harder to measure it so that most studies rely on a silent agreement between author and reader regarding the definition of stress. Furthermore, the impact of stress on memory is not always negative; it actually can help us by focusing our attention on a certain event, often improving our performances like before a university examination. Noteworthy, in our study,2 stress acted as a trigger in the majority of our patients with TGA; in fact, 54% had a “positive” (eg, sexual intercourse) or “negative” (eg, quarrel) stress just before TGA onset, yet we have emphasized that most of the trigger events are quite common so that their detection should not be considered a surprise when carefully interviewing patients with TGA. Lastly, it remains difficult to explain why such usual activities are not able to continuously replicate TGA symptoms during a patient’s life.

With regard to the ongoing study by Griebe et al on the role of stress in TGA, the authors will have the very tough task of clearly defining stress and giving methodological details on its measurement. It would also be very helpful to distinguish between physiological stress (positive impact on memory) and pathological stress (negative impact on memory) and give a threshold measure. Likewise, it is critical to distinguish between acute and chronic stress; is there a difference in terms of activation of the hypothalamic–pituitary–adrenal axis? We already know that chronic stress induces an increase of glucocorticoid hormone levels,3 but the mere presence of a neuroendocrine response is not sufficient to label it as stress nor is it indicative of the presence of a stressor.4 Finally, stress might just be associated and not directly correlated to TGA, as shown for jugular valve insufficiency in our study; stress might amplify the effect of hippocampal ischemia as shown in rats, but it does not explain the ischemia on its own.

In conclusion, Griebe et al will have to address all of these issues and we surely look forward to reading the results of their study. Indeed medicine, different from religion, does not rely solely on faith, but mainly on measurable and replicable data.5

Disclosures

None.

Claudio Baracchini, MD
Department of Neurological Sciences
University of Padua School of Medicine
Padova, Italy

Enzo Ballotta, MD
Vascular Surgery Section
Geriatric Surgery Clinic
Department of Surgical and Gastroenterological Sciences
University of Padua
School of Medicine
Padova, Italy

Renzo Manara, MD
Department of Neurological Sciences
University of Padua School of Medicine
Padova, Italy

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Claudio Baracchini, Enzo Ballotta and Renzo Manara

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