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Commentary

Refusing to imagine? On the possibility of psychogenic aphantasia. A commentary on Zeman et al. (2015)

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Our ability of “seeing” mental images in the absence of appropriate sensory input has always raised great interest not only in science, but also in philosophy (Sartre, 1940), art and literature (de Vito, 2012). Remarkably, however, some people claim not to experience visual mental images at all. Although the existence of these cases has been known to science for well over 100 years (Galton, 1880, 1883), the phenomenon has been oddly ignored and a systematic research is still lacking. Zeman, Dewar, and Della Sala (2015) provide important insights into several aspects of the lifelong inability to mentally visualize absent objects and label this condition “aphantasia”, a convenient term that may help focusing research on the phenomenon. The authors report on otherwise healthy people. However, this condition has been also observed in different populations of patients. Since Charcot (see Bartolomeo, 2008, for an English summary; Charcot & Bernard, 1883) neurologists have described cases of acquired inability to form visual mental images (review in Bartolomeo, 2002). However, it is worth noticing that in some of these cases, including the seminal Charcot case (Charcot & Bernard, 1883), an “abrupt and isolated” loss of visual imagery has been interpreted by some as having a psychogenic, rather than organic, origin (Zago et al., 2011). When considering evidence such as that reported by Zeman et al. (2015), it is important to keep this possibility in mind. Indeed, the case of imagery loss could engender an “organic/functional” debate analogous to the one that has revolved around

retrograde amnesia (e.g., De Renzi, Lucchelli, Muggia, & Spinnler, 1997).

Published in 1883 in *Le Progrès Médical*, the case of Monsieur X was presented as a sudden loss of the ability to construct mental images linked to a hypothetical circumscribed cerebral lesion and represents a classical citation in studies discussing the loss of visual imagery (e.g., Zeman et al., 2010). Although no evidence was available about the etiology or locus of the lesion (there was no post-mortem examination), left temporal damage sparing the occipital cortex might be hypothesized, given the described reading problems in the absence of elementary visual impairment (Bartolomeo, 2008). This hypothesis is consistent with neuropsychological and neuro-imaging studies revealing that large networks of brain areas are engaged during visual mental imagery, probably reflecting top-down influences from frontal and parietal regions to the temporal lobe (e.g., Mechelli, Price, Friston, & Ishai, 2004). However, it is also important to consider that, at the onset of his imagery disorder, Monsieur X experienced something akin to mental alienation. Things around him appeared strange and new and he became anxious. As Monsieur X wrote to Charcot, “I observed a drastic change in my existence that obviously mirrored a remarkable change in my personality. Before I used to be emotional, enthusiastic with a prolific imagination; today I am calm, cold and I lost my imagination” (p. 570, own transl.). The Portuguese neurologist António de Sousa Magalhães e Lemos (1906) observed a similar case of

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visual imagery loss in a woman with an experience of recurring hysterical crises, other than depressive and anxious symptomatology. However, once again, an organic root (i.e., damage to the connectional networks between memories and “the spared center of mental images”, p. 27, own transl.) was hypothesized for the disease. Cotard was the first to emphasize the existence of an intimate association between depressive/anxious symptomatology and visual imagery loss: “We cannot prevent ourselves from thinking that the relation” between these two phenomena “goes beyond the fortuitous coincidence” (Cotard, 1884, p. 292, own transl.). His two patients, M.P... and M***, with severe anxious and depressive symptoms, became totally incapable to shape mental images, and M.P... also developed the Cotard delusion, and believed he was dead. According to Cotard (1884), the delusion of non-existence of the self was likely to be determined by a misinterpretation of the mental imagery loss, since this symptom is common in patients with chronic anxiety. Since then, a number of similar cases was observed where the loss of visual imagery accompanied depersonalization and derealisation, which are dissociative disorders often associated with anxiety and depression (e.g., Deny & Camus, 1905; Dugas, 1898). In 1898, the French philosopher Ludovic Dugas coined the term “depersonalization” to describe “a state in which there is the feeling or sensation that thoughts and acts elude the self and become strange; there is an alienation of personality” (Dugas & Moutier, 1911, p. 12). Dugas (1898) reported the case of patient M., whose awareness of having become incapable to mentally visualize an absent person marked the onset of his chronic depersonalization. In 1960, the Italian psychiatrist Gian Carlo Reda described four patients who had experienced a loss of visual mental imagery in the context of a complex medical history involving anxiety attacks, depressive states, and, above all, depersonalization and derealisation (“I only have words in my mind, I cannot see anything”, Reda, 1960, p. 109). Simeon et al. (2000) also described the disturbance in mental imagery as one of the factors subsuming depersonalization/derealisation. Thus, it may be suggested the existence of two categories of patients (i.e., organic and functional) who may experience impaired visual imagery, with different characteristics (e.g., Zago et al., 2011, highlighted that the emphatic complain of the loss of visual imagery is recurrent in functional patients, but rare in neurological patients). A third category may also be possible, where organic and functional factors overlap. In light of such considerations, a critical reexamination of Monsieur X’s psychopathological symptomatology provided Zago et al. (2011) with clues as to the possible alternative functional root of his deficit. In fact, Monsieur X presented some of the symptoms identified in functional aphantasia (e.g., anxiety, depression, depersonalization with derealisation). Moreover, Freud, who personally examined Monsieur X, suspected a neurosis and Young and van de Wal (1996) suggested a hysterical origin.

Therefore, an impairment of the “mind’s eye” may be linked to heterogeneous variables, which need to be properly kept into account in future investigations. Cases of “aphantasia” should receive not only neuropsychological and neuroimaging assessments, but also psychopathological examination.

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