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#3112 Title: Acquired Aphantasia in 88 cases: a preliminary report

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AIMS

For most of us, visual imagery is a fundamental feature of day-to-day subjective experience. It is thought to play multiple cognitive roles[1]. However, there is widespread variation in the subjective intensity of visual imagery, ranging from extreme vividness to complete absence. The term "aphantasia" was coined recently to describe the latter, which is usually lifelong. While rarer, cases of acquired aphantasia can provide mechanistic insight. Isolated cases have long been reported[2], with some attempts at theoretical synthesis[3,4]. We give a preliminary description of 88 such cases identified from among ~14,000 people contacting us in the wake of publicity surrounding Aphantasia.

METHODS

Cases were selected from individuals contacting us spontaneously reporting reduced or absent intensity of visual imagery. Contacts were asked to complete two measures of visual imagery, the Vividness of Visual Imagery Questionnaire (VVIQ) and Imagery Questionnaire (IQ)[5].

RESULTS

Cases were divided into those in with a strong probability of a neurological cause (n=39), a psychological cause (n=20) and those about which we cannot yet be confident (n=29). Functional aphantasia appears likely to account for some of the cases in the third category. The commonest precipitating events were head injury (n=19), affective disorder (n=17) and stroke (n=13). Other causes included surgery (postoperative) (n=7), drugs (n=6), infectious or inflammatory disease (n=3), neurodegenerative disease (n=3), and seizure disorders (n=2). For subjects completing the VVIQ (n=29), the mean score was 20.1/80 (range 16-32,) indicating marked reduction of imagery vividness. Localizable lesions were predominantly right sided (n=6) and occurred in posterior cortical areas, particularly occipital and parietal, as well as two cases associated with damage to temporal cortex. Some cases reported other impairments, including impaired memory (n=12), prosopagnosia (n=5) and navigational difficulties (n=2). Of cases who reported on their dreams (n=28), around half had lost visual dreaming, a third had preserved visual dreaming and the remainder had visual dreaming of reduced intensity.

CONCLUSIONS

To our knowledge, this is the largest reported case series of acquired loss of visual imagery. Both neurological and psychological disorders can be responsible for acquired aphantasia. Our series includes cases of probable 'functional' aphantasia. Further detailed analysis of these cases is required.

1. Pearson. *Nat. Rev. Neurosci.* **20**, 624–634 (2019).
2. Zago *et al. Cognit. Neuropsychiatry* **16**, 481–504 (2011).

3. Farah. *Cognition* **18**, 245–272 (1984).
4. Bartolomeo. *Cortex* **38**, 357–378 (2002).
5. Zeman *et al. Cortex* **130**, 426–440 (2020).