Acquired Aphantasia in 88 cases: a preliminary report

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Introduction

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)[5,6].

Data was extracted from the free text of the original contact, any supplementary information provided such as medical records, and where available the responses to the IQ. We recorded basic demographic information, the precipitant of visual imagery loss, past medical history, the modality of imagery loss (visual or otherwise), and any comment on other impairments including the presence or absence of visual dreaming, changes to memory and changes to visual recognition.

Results

29 contacts completed the VVIQ and IQ, with a mean VVIQ score of 20.1/80 (range 16-32) indicating marked reduction of imagery vividness.

Contacts reported a wide variety of precipitating events for their visual loss; the commonest were head injury (n=21) and ischaemic damage (n=12) (figure 1). Based on the information received about the precipitating event and other medical background, cases were divided into those in with a strong probability of a neurological cause (n=39), a psychological cause (n=17) and those about which we cannot yet be confident (n=32 (figure 2). Functional aphantasia appears likely to account for some of the cases in the third category.

Available investigations enabled us to localise the lesion in 8 cases. These 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.

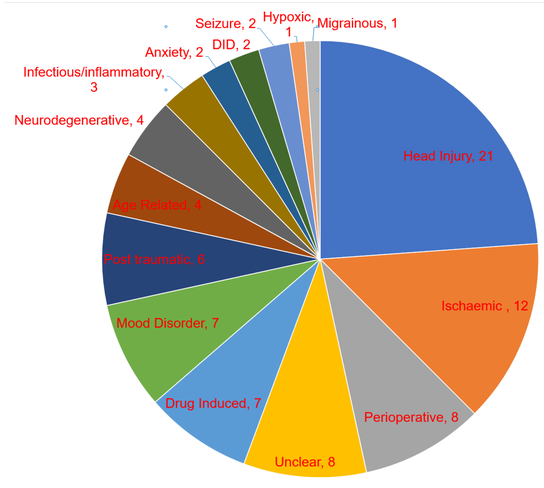

Figure 1. Precipitants of acquired aphantasia in our cohort.

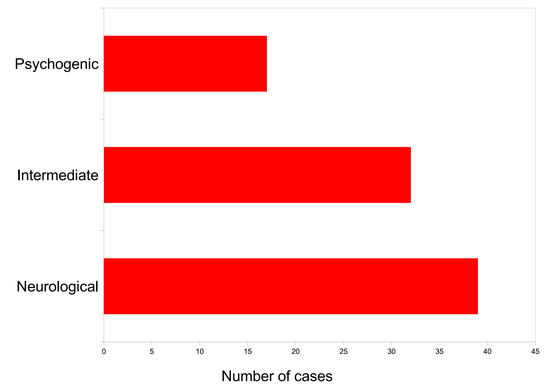

Figure 2. Broad classification of the underlying causes of acquired aphantasia.

Note that the classification above did not depend solely upon the reported precipitating cause of aphantasia as shown in figure 1, but on the author’s assessment of the totality of the available information. For example, cases following head injury were largely classified as intermediate, but 5 were classified as neurological based on other factors (for example MRI evidence of traumatic brain injury).

Conclusion

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.

References

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