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Reflections on aphantasia

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We recently coined the term ‘aphantasia’ to describe the experience of people who lack a ‘mind’s eye’ (Zeman, Dewar, & Della Sala, 2015), highlighting the features of lifelong aphantasia in a group of 21 individuals who had contacted us after reading a popular account of a related paper (Zeman, Della Sala, Torrens, Gountouna, McGonigle, & Logie, 2010) by Carl Zimmer in the science magazine *Discover* (Zimmer, 2010). Several consistent characteristics emerged: most participants discovered in their ‘teens or twenties, to their surprise, that when others spoke of ‘visualising’ items in their absence, these others had a genuinely ‘visual’ experience; the majority of our participants, in fact, had some experience of visual imagery from visual dreams or from involuntary ‘flashes’ of imagery occurring, for example, at sleep onset: their aphantasia thus involved a deficiency of voluntary imagery rather than a total absence; in general, the emotional consequences of discovering their aphantasia were modest; most participants regarded their autobiographical memory as poor.

In their commentary *‘Refusing to imagine? On the possibility of psychogenic aphantasia’*, de Vito and Bartolomeo draw attention to the possibility that in some cases at least, aphantasia may have a ‘psychological’ or ‘functional’ basis. The title of the commentary, ‘refusing to imagine’, suggests that the psychological process might involve an element of choice. We agree with these authors that ‘psychological’ factors such as mood can influence imagery, and that a full assessment of aphantasia should include a ‘psychopathological examination’. We also agree that almost every clinical phenomenon exists in a ‘factitious’ form. However, their commentary provokes additional thoughts.

De Vito and Bartolomeo allude to the traditional distinction between ‘functional’ and ‘organic’, referring specifically to the debate concerning the aetiology of retrograde amnesia (for contrasting views see De Renzi, 2002 and Kopelman, 2002). There is undoubtedly a useful working distinction between irreversible, structural causes of neuropsychological symptoms and reversible, functional ones. However the familiar distinction between the ‘functional’ and ‘organic’ is a dubious one: we are all organisms, and our functions and dysfunctions are therefore, necessarily, organic. The

neurologist Kinnier Wilson stated this point in especially uncompromising terms in a famous passage in his textbook: the ‘antithesis between ‘organic’ and ‘functional’ disease states ... lingers at the bedside and in medical literature, though it is transparently false and has been abandoned long since by all contemplative minds’ (Kinnier Wilson, 1940). We suspect that De Vito and Bartolomeo would agree. We labour the point here because it suggests the interesting hypothesis – echoed in discussions of retrograde amnesia (Lucchelli & Spinnler, 2002), as de Vito and Bartolomeo point out – that aphantasia will involve some common pathways in the brain whether it is due to reversible, ‘psychological’ or irreversible, ‘structural’ causes.

De Vito and Bartolomeo draw attention to controversy surrounding Charcot’s famous case of aphantasia, the case of Monsieur X (Charcot, 1889). In the absence of brain imaging data or post mortem findings, the aetiology of Monsieur X’s loss of imagery will never be known with certainty. It is true, as de Vito and Bartolomeo point out, that Monsieur X had been under unusual stress prior to the onset of his symptoms, and had fallen prey to uncharacteristic anxiety and low mood. These features could set the scene for ‘psychogenic aphantasia’. Several aspects of this complex case, however, point to a neurological basis for his aphantasia, including the abrupt onset of Monsieur X’s symptoms, their association with prosopagnosia and some disturbance of colour vision. Moreover his symptoms persisted despite an apparently successful readjustment to his new mental circumstances: ‘He realised, moreover, little by little, that he could by other means, by invoking the aid of other forms of memory, continue to successfully direct his business affairs. And thus, at the present time, he has become reconciled to the new situation...’ (Charcot, 1889, page 154). While Zago et al (Zago et al., 2011) argue for a psychological reading of Monsieur X’s case, Young and van de Wal (Young & van de Wal, 1996), contrary to de Vito and Bartolomeo’s account, underline the remarkable degree to which Charcot’s lecture anticipates the themes of subsequent descriptions of aphantasia. They do note with surprise, however, that Charcot failed even to consider the possibility of a psychological explanation in this case, given his interest in hysteria.

The data we presented from our series of 21 participants were derived from a questionnaire survey and lacked the systematic, psychological detail we hope to include in a future face-to-face study. However, several considerations suggest that these individuals' aphantasia is unlikely to have a primarily 'psychopathological' explanation. All participants described a characteristic of their mental lives that was, so far as they knew, lifelong. Aphantasia was a stable state rather than a variable trait. Few described any major emotional concomitants. Most were leading fulfilling personal and professional lives. We are impressed by one further association: impoverishment of imagery appears to be common in congenital prosopagnosia (Gruter et al., 2009). While our 21 participants did not specifically report problems with face recognition, it is likely that there is some overlap between aphantasia and congenital prosopagnosia, a condition few would regard as predominantly 'psychogenic'.

The vividness of self-reported visual imagery varies widely among healthy individuals (McKelvie 1995). In addition to this normal variation, the existing literature points to a range of pathological and pharmacological factors that can influence vividness. It can be dimmed or abolished by brain injury (Farah, 1984). Depression, anxiety and depersonalisation can have similar effects (Sierra, 2009), whereas imagery is heightened in psychopathologies such as post-traumatic stress disorder and by hallucinogens like mescaline and LSD (Carhart-Harris et al., 2012). In all these cases the precision of self-report is open to question, but the vagaries of introspection are unlikely to account entirely for the wide range of variation described both within and between individuals.

Thus, in essence, we fully agree with de Vito and Bartolomeo that psychological and psychiatric factors should be taken into account in the assessment of a person complaining of aphantasia. These factors will themselves have neural correlates. We are doubtful that psychopathology plays a major role among people reporting lifelong aphantasia.

History sometimes repeats itself: a further account by Carl Zimmer of our recent paper, in the New York Times (Zimmer, 2015), has prompted around a thousand individuals with aphantasia to make

contact with us, attesting to the popular interest and relative obscurity of this fascinating variation of human experience. We look forward to future explorations of the experiential, neuropsychological, neuropsychiatric and neural features of aphantasia.

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