



Heriot-Watt University  
Research Gateway

## Reflections on aphantasia

**Citation for published version:**

Zeman, AZJ, Dewar, MT & Della Sala, S 2016, 'Reflections on aphantasia', *Cortex*, vol. 74, pp. 336-337.  
<https://doi.org/10.1016/j.cortex.2015.08.015>

**Digital Object Identifier (DOI):**

[10.1016/j.cortex.2015.08.015](https://doi.org/10.1016/j.cortex.2015.08.015)

**Link:**

[Link to publication record in Heriot-Watt Research Portal](#)

**Document Version:**

Peer reviewed version

**Published In:**

Cortex

**General rights**

Copyright for the publications made accessible via Heriot-Watt Research Portal is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

**Take down policy**

Heriot-Watt University has made every reasonable effort to ensure that the content in Heriot-Watt Research Portal complies with UK legislation. If you believe that the public display of this file breaches copyright please contact [open.access@hw.ac.uk](mailto:open.access@hw.ac.uk) providing details, and we will remove access to the work immediately and investigate your claim.

# Accepted Manuscript

Reflections on aphantasia

Adam Zeman, Professor of Cognitive and Behavioural Neurology, Michaela Dewar, Assistant Professor and Research Leader, Sergio Della Sala, Professor of Human Cognitive Neuroscience



PII: S0010-9452(15)00309-3

DOI: [10.1016/j.cortex.2015.08.015](https://doi.org/10.1016/j.cortex.2015.08.015)

Reference: CORTEX 1577

To appear in: *Cortex*

Received Date: 10 August 2015

Revised Date: 14 August 2015

Accepted Date: 14 August 2015

Please cite this article as: Zeman A, Dewar M, Della Sala S, Reflections on aphantasia, *CORTEX* (2015), doi: [10.1016/j.cortex.2015.08.015](https://doi.org/10.1016/j.cortex.2015.08.015).

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

## Reflections on aphantasia

Adam Zeman<sup>a</sup>, Michaela Dewar<sup>b</sup>, Sergio Della Sala<sup>c</sup>

<sup>a</sup> Professor of Cognitive and Behavioural Neurology, University of Exeter Medical School, College House, St Luke's Campus, Exeter EX1 2LU (corresponding author)

[a.zeman@exeter.ac.uk](mailto:a.zeman@exeter.ac.uk)

<sup>b</sup> Assistant Professor and Research Leader, Psychology, School of Life Sciences, Heriot-Watt University, Edinburgh EH14 4AS

[m.dewar@hw.ac.uk](mailto:m.dewar@hw.ac.uk)

<sup>c</sup> Professor of Human Cognitive Neuroscience, Human Cognitive Neuroscience, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ

[sergio@ed.ac.uk](mailto:sergio@ed.ac.uk)

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.

ACCEPTED MANUSCRIPT

## References

Carhart-Harris, R. L., Erritzoe, D., Williams, T., Stone, J. M., Reed, L. J., Colasanti, A. et al. (2012). Neural correlates of the psychedelic state as determined by fMRI studies with psilocybin. *Proc.Natl.Acad.Sci.U.S.A*, *109*, 2138-2143.

Charcot, J.M. (1889). *Clinical Lectures on Diseases of the Nervous System*. (vols. 3) London: The New Sydenham Society.

De Renzi, E. (2002). What does psychogen mean? *Cortex*, *38*, 678-681.

Farah, M.J. (1984). The neurological basis of mental imagery: a componential analysis. *Cognition*, *18*, 245-272.

Gruter, T., Gruter, M., Bell, V., & Carbon, C. C. (2009). Visual mental imagery in congenital prosopagnosia. *Neurosci.Lett.*, *453*, 135-140.

Kinnier Wilson, S.A. (1940). *Neurology* (Vol 2). New York: Hafner Pub Co Inc.

Kopelman, M. D. (2002). Organic retrograde amnesia. *Cortex*, *38*, 655-659.

Lucchelli, F. & Spinnler, H. (2002). The "psychogenic" versus "organic" conundrum of pure retrograde amnesia: is it still worth pursuing? *Cortex*, *38*, 665-669.

McKelvie S. (1995). The VVIQ as a psychometric test of individual differences in visual imagery vividness: a critical quantitative review and plea for direction. *Journal of Mental Imagery*, *19*, 1-106,.

Sierra, M. (2009). *Depersonalisation: a new look at a neglected syndrome*. Cambridge: Cambridge University Press.



Young, A. & van de Wal, C. (1996). Charcot's Case of Impaired Imagery. In Code C, Wallesch C-W, Y. Joannette, & A. Roch Lecours (Eds.), *Classic Cases in Neuropsychology* (pp. 31-44). Hove, East Sussex: Psychology Press.

Zago, S., Allegri, N., Cristoffanini, M., Ferrucci, R., Porta, M., & Priori, A. (2011). Is the Charcot and Bernard case (1883) of loss of visual imagery really based on neurological impairment? *Cogn Neuropsychiatry*, *16*, 481-504.

Zeman, A., Dewar, M., & Della Sala, S. (2015). Lives without imagery - Congenital aphantasia. *Cortex*, <http://dx.doi.org/10.1016/j.cortex.2015.05.019>

Zeman, A.Z., Della Sala, S., Torrens, L.A., Gountouna, V.E., McGonigle, D.J. & Logie, R.H. (2010). Loss of imagery phenomenology with intact visuo-spatial task performance: a case of 'blind imagination'. *Neuropsychologia*, *48*, 145-155.

Zimmer, C. (2010). The Brain. *Discover*, 28-29.

Zimmer C. (2015) Picture this? Some just can't. *New York Times*, June 23<sup>rd</sup>, D3.