Reality monitoring and visual hallucinations in Parkinson’s disease

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Abstract

Between 8 and 40% of Parkinson disease (PD) patients will have visual hallucinations (VHs) during the course of their illness. Although cognitive impairment has been identified as a risk factor for hallucinations, more specific neuropsychological deficits underlying such phenomena have not been established. Research in psychopathology has converged to suggest that hallucinations are associated with confusion between internal representations of events and real events (i.e. impaired-source monitoring). We evaluated three groups: 17 Parkinson’s patients with visual hallucinations, 20 Parkinson’s patients without hallucinations and 20 age-matched controls, using tests of visual imagery, visual perception and memory, including tests of source monitoring and recollective experience. The study revealed that Parkinson’s patients with hallucinations appear to have intact visual imagery processes and spatial perception. However, there were impairments in object perception and recognition memory, and poor recollection of the encoding episode in comparison to both non-hallucinating Parkinson’s patients and healthy controls. Errors were especially likely to occur when encoding and retrieval cues were in different modalities. The findings raise the possibility that visual hallucinations in Parkinson’s patients could stem from a combination of faulty perceptual processing of environmental stimuli, and less detailed recollection of experience combined with intact image generation.

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1. Background

Psychiatric disturbance in Parkinson’s disease (PD) particularly visual hallucinations (VHs) [26,57] can be a frequent and disturbing complication of the disease. There have been several recent surveys, which give prevalence estimates of VHs between 9.8 and 44% [33,66,31,1]. Furthermore, VHs have been reported with all anti-Parkinson’s medication [14,72] and it is estimated that up to 33% of PD patients undergoing long-term treatment will have visual hallucinations during the course of their illness [66,14,72] (see Barnes and David [4] for systematic review). Nevertheless, comparisons of hallucinators and non-hallucinators have seldom shown major differences in drug history [66] and VHs have been reported in PD patients before any drugs have been taken [62]. Factors that were highlighted in Barnes and David’s review which predispose PD patients to visual hallucinations included length of illness, increased age and cognitive decline. In addition, psychosis, sleep disturbances, depression and certain personality profiles have been implicated. However, the precise neuropsychological underpinnings for VHs in this group have yet to be delineated (for a neurological view see Manford and Andermann [49]).

From the psychopathology literature several lines of evidence have emerged which suggest that hallucinations are linked to a bias towards attributing to an external source, an event which had been generated by oneself—a problem with source or reality monitoring [7,42]. This could entail the perception of inner thoughts as external voices [21,29,52]. Indeed, auditory hallucinators seem to have a bias towards attributing their own words to another person when compared with either psychiatric or normal controls [7,56]. In terms of visual hallucinations the general hypothesis would be that a failure in reality monitoring leads to a confusion between self-generated mental images and perception.

Visual hallucinators may not only confuse imagination with perception, but the products of previously imagined material may be confused with products of previously perceived material [13,42]. According to the source-monitoring framework [44], several factors are involved in the attribution of mental experiences to sources, for example, their qualitative features (e.g. familiarity and more specific information...
such as perceptual and emotional detail), and the criteria one adopts for evaluating such information (e.g. motives, beliefs etc.) Especially important for accurate source attributions is the accurate recollection of specific details. That is, typically, familiarity alone is not a particularly reliable cue to the origin of information. Thus, the less recollection one has of an encoding event, the more that memory is susceptible to misinterpretation, particularly as regards source, hence the risk of subsequent confusion between imaginary and real events. It is known that normal ageing alone can cause deficits in source monitoring \cite{17, 23, 27} and an over reliance on familiarity \cite{59–61}. Accentuation of these deficits plus the addition of others may well be relevant to the susceptibility to VHs in Parkinson’s disease.

Another general risk factor for hallucinations, VHs in particular, is sensory impairment \cite{37}. This is most obvious in the Charles Bonnet syndrome which comprises florid realistic hallucinations—similar phenomenologically to those seen in PD \cite{4} and commonly associated with late acquired visual deficits \cite{69, 73}. Such deficits whether they occur peripherally to centrally may each contribute to VHs \cite{3, 16, 49} by disinhibiting or “releasing” endogenous imagery processes \cite{68}. In the current study of Parkinson’s patients, we hypothesised that a combination of visual processing deficits \cite{19, 76} and episodic memory impairment, both found in this group \cite{67}, combined with source-monitoring errors, could underlie the propensity to experiencing VHs. In order to test this, we studied PD patients with and without VHs, and age-matched healthy controls. We predicted that there would be differences between the PD group as a whole and the controls in terms of generalised cognitive impairment, but that the hallucinators would show specific deficits in object perception and source monitoring. Furthermore, we argued that for the positive phenomena of hallucinations (perception without an external object \cite{2}), image generation or visual imagery processes would have to be intact.

2. Methods

2.1. Participants

Participants were recruited through a questionnaire study carried out at the University of Reading, via local branches of the UK Parkinson’s Disease Society, and at a neurology clinic at King’s College Hospital. All patients had a presumptive clinical diagnosis of PD. Individuals were assigned to groups according to whether they had experienced VHs in the last 3 months, and those who had never experienced VHs. No patient in the population sampled had a clinical diagnosis of either Alzheimer’s disease or Lewy body dementia. Those indicating problems were asked to complete a detailed hallucinations questionnaire and were invited to attend a clinical and neuropsychological assessment. The questionnaire comprised items covering the nature and properties of the subject’s visual hallucinations \cite{4}. Patients with eye disease or migraine or other concurrent medical conditions were excluded. Duration of illness and medication were recorded and stage of illness was scored according to the Hoehn and Yahr scale \cite{36}.

2.2. Design

The study consisted of two sections. In the first, participants were evaluated on a range of standard neuropsychological tests. Following this, imagery, recognition memory and source memory were assessed with a picture/word recognition task.

2.3. Neuropsychological tests

A battery of neuropsychological tests was administered. This included the visual object and space perception battery (VOSP) to assess visual perception ability \cite{75}. This comprises 4 spatial sub-tests: dot counting, position discrimination, number location and cube analysis, and 4 object sub-tests: incomplete letters, silhouettes, object decision and progressive silhouettes (see Figs. 1–3). Memory ability was measured by a recognition memory test for faces and words \cite{74}, which has a two-alternative forced choice format. Visual imagery ability was measured by structured imagery questions similar to those detailed in Farah et al. \cite{24}. In brief, 10 questions were asked, requiring one-word answers on each of the following topics: shapes, letters and numbers. The questions were designed such that mental imagery would be used to provide the answers. For example: “A sofa is taller than it is wide” (true or false?). Is the capital letter Q formed by straight lines, curved lines or both? Is a raspberry darker red than a strawberry? In addition 4 sets of 10 semantic decision questions were asked concerning living and nonliving items, both visual and non visual. For example: living-visual—Does a giraffe have small horns? Living non-visual—Are frogs amphibians? Non-living visual—Is a teacup taller than a mug? Non-living non-visual—Is a kettle used for baking? The vividness of visual imagery questionnaire (VVIQ) \cite{51} was used as a standardised self-report rating of subjective imagery. This has 16 items, which are scored on a 5-point scale (1—perfectly clear, to 5—no image at all). Cognitive flexibility was measured by the verbal fluency test (FAS) \cite{10}. Depressed mood was measured with the 21-item Beck Depression Inventory \cite{5}. The mini mental state examination (MMSE) \cite{28}, was utilised as a general cognitive screening test and the national adult reading test (NART) \cite{58} as an estimate of pre-morbid IQ.

2.4. Experimental memory and source task

The stimuli for this task consisted of two packs of 120 mm × 80 mm cards with one stimulus on each card taken from the Snodgrass and Vanderwart \cite{71} set of standardised pictures. The encoding pack consisted of 24 words.
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