Hallucinogen-persisting perception disorder

Leo Hermle, Melanie Simon, Martin Ruchsow and Martin Geppert

Therapeutic Advances in Psychopharmacology 2012 2: 199 originally published online 17 June 2012
DOI: 10.1177/2045125312451270

Hallucinogen-persisting perception disorder

Ther Adv Psychopharmacol
(2012) 2(5) 199–205
DOI: 10.1177/2045125312451270

Leo Hermle, Melanie Simon, Martin Ruchsow and Martin Geppert

Abstract: A 33-year-old female patient developed a hallucinogen-persisting perception disorder (HPPD) after lysergic acid diethylamide (LSD) abuse for a year at the age of 18. Specifically, she reported after images, perception of movement in her peripheral visual fields, blurring of small patterns, halo effects, and macro- and micropsia. Previous treatment with antidepressants and risperidone failed to ameliorate these symptoms. Upon commencing drug therapy with lamotrigine, these complex visual disturbances receded almost completely. Based on its hypothesized neuroprotective and mood-stabilizing effects, the antiepileptic lamotrigine may offer a promising new approach in the treatment of HPPD.

Keywords: Flashback, hallucinogen-persisting perception disorder, LSD

Correspondence to:
Martin Ruchsow, MD
Department of Psychiatry, Christophsbad, Faurndauer Str. 6 – 28, 73035 Göppingen, Germany
martin.ruchsow@christophsbad.de

Leo Hermle, MD
Melanie Simon, MA
Martin Geppert, MD
Department of Psychiatry, Christophsbad, Göppingen, Germany

Introduction

The complex phenomenology of acute hallucinogen-induced psychosis has been described and analysed extensively over the years. However, the clinical relevance of the long-term psychological sequelae which include so-called flashbacks remains unclear [Hermle *et al.* 1992; Hermle *et al.* 2008]. Moreover, a consistent etiological model to explain these effects has yet to be proposed. Ever since the first description [Cooper, 1955], reports about the incidence of post-toxic flashbacks show a wide variation. Between 5% and 50% of hallucinogen users are reported to have experienced at least one flashback [Alarcon *et al.* 1982; McGee, 1984].

Flashbacks, echo phenomena and other psychotic manifestations typically occur after drug-free periods of varying lengths. In the International Classification of Diseases, 10th revision (ICD-10), such hallucinogen-induced echo psychoses are listed under F16.70 [Dilling *et al.* 1991; Pechnik and Ungerleider, 2004]. However, the *Diagnostic and Statistical Manual of Mental Disorders*, fourth revised edition (DSM-IV-R) [American Psychiatric Association, 1994], classifies these phenomena under the term 'hallucinogen persisting perception disorder (HPPD)' – defined as a long-lasting condition characterized by spontaneous recurrence of visual disturbances reminiscent of acute hallucinogen intoxication. Such experiences may take the form of various geometric shapes, objects in the peripheral visual fields, flashes of different colours, enhanced colour intensity, trailing and stroboscopic perception of moving objects, after images, halos and macro- and micropsia. Furthermore, these episodes may persist for years. At variance with DSM-IV-R, ICD-10 recognizes hallucinogen-induced visual disturbances as lasting only seconds to minutes.

It is important to note that in contrast to classical psychotic disorders, patients with HPPD recognize the unreal nature of their visual disturbances which qualifies them as pseudohallucinations.

Method

A MEDLINE literature search (1994–2011) with the keywords 'Hallucinogen persisting perception disorder HPPD' was conducted.

Case report

History

The female patient, now 33 years old and an architect by profession, reported the recreational use of up to 30 doses of lysergic acid diethylamide (LSD; 'tabs') during a 1-year stay in the USA at the age of 18. Each single dose was probably limited to 100 µg and consumed in a peer group setting. She also used marijuana for relaxation and occasionally experimented with ecstasy, psilocybin mushrooms and ketamine.

Approximately 2–3 weeks after returning to Europe, and the last drug taking, the patient developed persistent visual disturbances from which she has been suffering ever since. She described these as attenuated 'flashbacks' – comparable with the experiences during acute LSD intoxication. More specifically, the patient mentioned the occurrence of after images, the perception of motion in the periphery of her visual fields, flickering when looking at patterned objects, halo effects, macro- and micropsia, and in the patient's own words, 'a glow-worm effect' meaning the perception of bright little spots of light across the visual field. With her eyes shut, no such abnormalities were perceived.

These symptoms persisted for the last 13 years, with little change in intensity and frequency. All efforts at treatment, psychopharmacological as well as psychotherapeutic, failed to alleviate the symptoms. Often the patient was unable to focus properly with her eyes and tired rapidly while performing intense visual tasks – these deficiencies being detrimental to her studies and professional work as an architect. As a consequence, the patient became depressed with latent suicidal impulses. She also found it increasingly difficult to distinguish between 'normal' and ' abnormal' perceptions.

Earlier in 2011, the patient underwent an 8-week course of psychosomatic treatment for depression as an outpatient at a university hospital clinic in southern Germany. Despite a significant improvement in her mood, the remission was only partially leading to a low-level continuous depression classified as dysthymia.

From 2006 to 2008 the patient received fixed doses of sertraline (200 mg/day) for 13 months, citaloprame (20–30 mg/day) for 6 months and fluoxetine (20 mg/day) for 5 months. These selective serotonin reuptake inhibitors (SSRIs) alleviated depression but did not relieve the HPPD symptoms. Due to weight gain the SSRIs were discontinued in September 2008. In October 2008 she was prescribed 0.5–1.0 mg risperidone without any effect. The medication was discontinued after 6 weeks.

Treatment course

Following informed consent, a trial of the antiepileptic lamotrigine was initiated to combat the unrelenting visual disturbances of the patient. With regular drug therapy over at least 12 months (maximum dose 200 mg of lamotrigine for 6 months, presently 100 mg), some of the abnormal perceptions such as 'sense of levitation' or macro-/micropsia disappeared completely whereas a qualitative improvement was noted with other symptoms (sense of motion of stationary objects, flickering etc.). The 'sense of levitation' indicates that this case of HPPD was more complex as it included more than just visual abnormalities. Furthermore, after images, halos, and 'glow worm' effects occurred less frequently. Rapid improvement was registered even during the dosing-in phase of lamotrigine – before the administration of therapeutic doses. Addition of SSRI-type antidepressants to the drug regime did not yield any beneficial effects. Instead they increased the frequency of derealization and depersonalization episodes in the patient. This was reversed to a large extent upon cessation of SSRI therapy. The patient also noted positive effects of psychotherapeutic intervention on attention focusing and mood stabilization. Not surprisingly, increased overall stress levels correlated with worsening of her symptoms.

Neuropsychological testing

As part of the initial psychiatric assessment, extensive neuropsychological profiling was undertaken (Table 1). Memory functions, attention span, visuo construction and frontal-executive functions were examined. No significant cognitive deficits were detected, except for underperformance in the test for phasic attention. With respect to her mental wellbeing, the patient's self assessment indicated a light to medium depressive and anxiety disorder, most likely attributable to the chronic distress resulting from the abnormal perceptions. She had low self esteem, was emotionally unstable and introverted.

A repeat assessment 19 months later showed a continued improvement of her already normal to above average attention performance which was at odds with the patient's subjective perception of diminished concentration faculties in everyday life. No change was observed in her mental state, with a persistent low to medium depression indicative of a dysthymic disorder (Beck Depression Inventory IV, Self-report Symptom Inventory 90 Items – Revised). Thus, the serotonergic antidepressants administered during this period proved largely ineffective. However, the patient noticed a reduction in her anxiety and phobias that was also

Table 1. Psychological tests.

		Initial evaluation (March 2010)	Follow-up evaluation (October 2011)
Cognitive performances			
Attention functions			
Sustained attention (DAUF)	finished stimuli	PR 90	
	errors	PR 82	
	reaction time	PR 91	
Alertness (TAP)	without warning tone	PR 10	PR 92
	without warning tone	PR 5	PR 95
Divided attention (TAP)		PR 42	PR 46
Go/no go (TAP)		PR 73	PR 82
Coding (WAIS)		score 12	
Executive functions			
Flexibility (TAP)	verbal	PR 86	
	nonverbal	PR 79	
Tower of London		PR 52	
Memory			
VLMT	learning performance immediate	PR 80	
	recall delayed	PR 45–65	
	recall	PR 25–45	
	recognition	PR >65	
Digit span (WAIS)		score 12	
Rey-figure	delayed recall	PR 84	
Visual perception			
VOSP		normal	
Rey-figure	copy	PR >99	
Sensitivities (only conspicuous findings)			
BDI-II		score 20	score 19
SCL-90R	depression	T 64	T 65
	obsessive–compulsive	T 65	T 70
	interpersonal sensitivity	normal	T 60
	phobic anxiety	T 70	T 62
	Psychoticism	T 64	T 61
	GSI	T 62	T 60
	PSDI	T 61	T 61
	PST	Normal	Normal
Personality (only conspicuous findings)			
SCID-II	APD	below cutoff	6
	OCPD	5	below cutoff
	BPD	6	7
	ASPD	4	below cutoff
FPI-R	satisfaction with oneself	stanine 1	
	need for achievement	stanine 1	
	shyness	stanine 8	
	aggressiveness	stanine 3	
	health sorrows	stanine 1	
	extraversion	stanine 1	
	emotionality	stanine 8	

APD, avoidant personality disorder; ASPD, antisocial personality disorder; BDI-II, Beck Depression Inventory; BPD, borderline personality disorder; DAUF, Sustained Attention Test; FPI-R, Freiburg Personality Inventory – Revised; GSI, Global Severity Index; OCPD, obsessive–compulsive personality disorder; PR, percentile rank; PSDI, Positive Symptom Distress Index; PST, positive symptom total; SCID-II, Structured Clinical Interview II; SCL-90R, Self-report Symptom Inventory 90 Items – Revised; TAP, Testbattery of Attentional Performance; VLMT, Verbal Learn And Memory Test; VOSP, Visual Object and Space Perception Battery; WAIS, Wechsler Adult Intelligence Test.

reflected in her psychological test scores. As for her personality, the patient's self esteem deteriorated even further during the 18-month observation period whereas obsessive–compulsive and anti-social traits receded.

Additional investigations

Results from brain magnetic resonance imaging scans, median nerve somatosensory evoked potentials, electroencephalograms and visual evoked potential tests were all normal.

Discussion

HPPD as defined in DSM-IV-R is a post-hallucinogen intoxication disorder encompassing a range of mostly visual perceptual disturbances that occur within a certain time frame after cessation of drug use. The definition of this disease entity is entirely based on studies involving chronic LSD users [Abraham, 1982, 1983]. However, any additional psychopathological symptoms that may have occurred in this patient population were not given any consideration. Pharmacotherapy of this very distressing condition is limited and any recommendations are based almost entirely on uncontrolled studies on small patient populations or even single case observations. Thus far, SSRIs, benzodiazepines, risperidone, olanzapine and naltrexone have all been tried with sometimes contradictory outcomes (Table 2).

Here we present the case of a 33-year-old female former LSD user who displayed typical symptoms of HPPD over an extended period of time. Concomitantly, she developed a depressive and anxiety disorder. Both perceptual and affective disorder proved largely unresponsive to various psychopharmacological interventions.

Lamotrigine is a widely used antiepileptic and mood-stabilizing drug which acts by blocking sodium and voltage-gated calcium channels and inhibiting glutamate-mediated excitatory neurotransmission. Additionally, there are data supporting a neuroprotective effect [Halonen *et al.* 2001]. Lamotrigine has also been shown to reduce symptoms of depersonalization and derealization [Sierra *et al.* 2001], although the same group was unable to reproduce their results in a placebo-controlled follow-up study [Sierra *et al.* 2003]. Given that excitotoxic destruction of inhibitory interneurons may play a role in at least some of the visual symptoms of HPPD (see below), lamotrigine was considered a possible treatment option for this patient. Furthermore, lamotrigine is generally well tolerated with a relative lack of adverse effects, making it a drug of choice for youths and young adults.

During a year-long trial of lamotrigine, with a maximum dose of 200 mg, the patient experienced significant relief from her symptoms, some of which disappeared completely. Only the depersonalization and derealization proved somewhat refractory. It is important to note that the patient showed a marked improvement during the 200 mg dosing-in phase itself and remained stable even after the dose was reduced to 100 mg daily.

To date, drug therapy for HPPD remains problematic. Abraham and colleagues hypothesized that flashbacks may have their pathophysiological basis in the excitotoxic destruction of inhibitory interneurons that carry serotonergic and GABAergic receptors on their cell bodies and terminals, respectively [Abraham *et al.* 1996]. Accordingly, benzodiazepines should be beneficial whereas atypical antipsychotics such as risperidone are expected to be detrimental to the symptoms of HPPD [Alcantara, 1998;Young, 1997]. In a case report of two patients diagnosed with post-LSD schizophrenia, administration of risperidone (3 mg daily) resulted in a transient occurrence of visual disturbances that disappeared completely with continued antipsychotic therapy [Lerner *et al.* 2002b]. In another open study that included eight patients diagnosed with HPPD, clonidine at a daily dose of 3 x 0.025 mg for 2 months led to a significant improvement of symptoms in six patients [Lerner *et al.* 2000]. SSRIs appear to worsen symptoms of HPPD, at least during the initial phase of treatment: People with HPPD treated with SSRIs and atypical antipsychotics (risperidone, olanzapine) reported an initial exacerbation of their flashbacks with a subsequent gradual improvement over time [Markel *et al.* 1994; Bonson *et al.* 1996; Espiard *et al.* 2005; Aldurra and Crayton, 2001]. It remains unclear whether this was due to the mood-enhancing effect with concomitant mental stabilization, or an increase in the diminished serotonergic neurotransmission in the visual cortex. Dramatic improvement with naltrexone (50 mg daily) was reported by Lerner and colleagues in two young men (aged 22 and 24 years) with LSD-induced HPPD. The remission was sustained as it was possible to discontinue the naltrexone after 2 months without precipitating a relapse [Lerner *et al.* 1997].

Table 2. Case reports on HPPD treatment.

Author (year)	Drug	Sample size	Study design	Major results
Abraham [1983]	Benzodiazepines Phenothiazines	21	Observational study	8 of the 9 subjects receiving benzodiazepines reported a reduced intensity and frequency of visual disturbances, whereas 11 of 12 subjects receiving phenothiazines reported exacerbation of HPPD
Abraham and Mamen [1996]	Risperidone	3	Case reports	3 patients with HPPD treated with risperidone reported an exacerbation of LSD-like panic and visual symptoms
Lerner *et al*. [1997]	Naltrexone	2	Case reports	Dramatic improvement with naltrexone (50 mg daily) was reported in two young men with LSD-induced HPPD. The remission was sustained as it was possible to discontinue the naltrexone after 2 months without precipitating a relapse
Lerner *et al*. [2000]	Clonidine	8	Observational study	6 of the 8 subjects (2 dropped out) received clonidine (0.025 mg, three times a day) for 2 months which alleviated LSD-related HPPD
Lerner *et al*. [2001]	Clonazepam	2	Case reports	2 outpatients with LSD-induced HPPD were successfully treated with clonazepam. They had not responded to low doses of classic antipsychotics or low-potency benzodiazepines
Lerner *et al*. [2002a]	Reboxetine	1	Case report	During a 6-month follow-up period on reboxetine (6mg/day) no exacerbations of visual disturbance were reported
Lerner *et al*. [2003]	Clonazepam	16	Observational study	16 patients received clonazepam 2 mg/day for 2 months. Patients reported significant relief during clonazepam administration.This improvement persisted during a 6-month follow-up period
Espiard *et al*. [2005]	Olanzapine Risperidone Sertraline	1	Case report	A case of a young man presenting with HPPD after a mixed intoxication with psilocybin and cannabis. Olanzapine (5 mg) exacerbated symptoms and was replaced by risperidone (2 mg/day) and sertraline (150 mg/day). After 6 months of this treatment HPPD disappeared

HPPD, hallucinogen-persisting perception disorder; LSD, lysergic acid diethylamide.

However, the 'success' of pharmacotherapy for HPPD should be treated with caution as this disorder appears to have a high propensity for spontaneous remissions – up to 50% of cases within a few months [Abraham, 2001]. In this context, the rarely ever documented occurrence of flashbacks in controlled studies of hallucinogen action should be mentioned. Apparently a favourable protective 'setting' may prevent the development of anxiety and psychotic decompensation as well as the loss of self control. In our case, a spontaneous remission coinciding with lamotrigine treatment appeared unlikely after a 13-year duration of unrelenting symptoms.

With a multitude of potential etiologies, it may not be possible to put forward a unified pathophysiological model of HPPD. Rather, a multifactorial origin of HPPD-related phenomena is to be assumed that may differ from case to case. The range of case-specific variables may extend from learning and kindling effects, individual reaction

patterns to mental trauma and weak self esteem to other psychophysic vulnerabilities [Hermle *et al.* 2008]. Additionally, only a small spectrum of hallucinogens seem capable of eliciting flashbacks, with LSD being the leading causative agent. In addition to the illicit nature of its use in an 'uncontrolled' environment, the long half life of LSD and the above-mentioned destabilizing effect on self realization may contribute to the relatively frequent development of flashbacks observed with this particular drug. Closely related to LSD in its psychotropic actions is psilocybin, which produces similar but shorter-lasting intoxications. Interestingly, there is only one documented case of HPPD following ingestion of *Psilocybe semilanceata* mushrooms in the psychiatric literature, despite its common use in the hippie subculture of the 1960s and 1970s [Espiard *et al.* 2005]. The incidence of mental disorders in 200 Native Americans of the Navajo tribe after ritual use of mescaline was the subject of a recent study by Halpern [Halpern, 2003]. Over a 3-year period of observation, not a single case of HPPD was detected.

The clinical relevance of flashbacks as sequelae of LSD and other biogenic and synthetic hallucinogens needs to be reassessed. In the light of more recent studies, earlier estimates of 5–54% incidence seem exaggerated – a rate of 5% or lower appears more realistic. With the Cochrane Society's strict criteria for evidence-based medicine as a yardstick, our current knowledge does not allow for any empirical recommendations as to the rational pharmacotherapy of HPPD. Future clinical research needs to be directed towards randomized controlled trials to establish sound treatment guidelines, in particular for chronic forms of HPPD [Halpern and Pope, 2003].

Implications for clinical practice

For clinical practice it is important to remember that first-generation 'classical' antipsychotics are not generally helpful in the treatment of persistent echo phenomena or HPPD (ICD-10 and DMS-IV-R, respectively). In fact a worsening of symptoms has been frequently reported. Other centrally acting drugs such as clonazepam, SSRIs, 'atypical' antipsychotics (e.g. risperidone, olanzapine), clonidine and naltrexone have been used with varying success, although not in randomized placebo-controlled clinical trials. Our own case indicates that the antiepileptic and mood stabilizer lamotrigine may offer a novel treatment for HPPD. Obviously, treatment of HPPD should also involve abstinence from all substances of abuse, stress reduction and treatment of comorbidities (depression, anxiety, and less often, psychosis).

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest statement

The authors declare no conflicts of interest in preparing this article.

References

Abraham, H. (1982) A chronic impairment of colour vision in users of LSD. *Br J Psychiatry* 140: 518–520.

Abraham, H. (1983) Visual phenomenology of the LSD flashback. *Arch Gen Psychiatry* 40: 884–889.

Abraham, H. (2001) New hope for hallucinogen-induced persistent perceptual disorder? (letter). *J Clin Psychopharmacol* 21: 344.

Abraham, H., Aldridge, A.M. and Gogia, P. (1996) The psychopharmacology of hallucinogens. *Neuropsychopharmacology* 14: 285–298.

Abraham, H. and Mamen, A. (1996) LSD-like panic from risperidone in post-LSD-visual disorder. *J Clin Psychopharmacol* 16: 238–241.

Alarcon, R., Dickinson, W. and Dohn, H. (1982) Flashback phenomena: clinical and diagnostic dilemmas. *J Nerv Ment Dis* 170: 217–223.

Alcantara, A. (1998) Is there a role for the alpha2 antagonism in the exacerbation of hallucinogen-persisting perception disorder with risperidone? *J Clin Psychopharmacol* 18: 487–488.

Aldurra, G. and Crayton, J. (2001) Improvement of hallucinogen persisting perception disorder by treatment with a combination of fluoxetine and olanzapine: case report. *J Clin Psychopharmacol* 21: 343–344.

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, 4th ed. Washington DC: American Psychiatric Press

Bonson, K., Buckholtz, J. and Murphy, D. (1996) Chronic administration of serotonergic antidepressants attenuates the subjective effects of LSD in humans. *Neuropsychopharmacology* 14: 425–436.

Cooper, H. (1955) Hallucinogenic drugs. *Lancet* 1: 1078–1079.

Dilling, H., Mombour, W. and Schmidt, M. (Eds) (1991) Mental and Behavioural Disorders (Including Disorders of Psychological Development). Clinical Descriptions and Diagnostic Guidelines. In: Tenth Revision of the International Classification of Diseases, chapter V (F): Bern: Huber (German Translation).

Espiard, M., Lecardeur, L., Abadie, P., Halbecq, I. and Dollfus, S. (2005) Hallucinogen persisting disorder after psilocybin consumption: a case study. *Eur Psychiatry* 20: 458–460.

Halonen, T., Nissinen, J. and Pitkänen, A. (2001) Effect of lamotigine treatment on status epilepticus-induced neuronal damage and memory impairment in rat. *Epilepsy Res* 46: 205–223.

Halpern, J. (2003) Hallucinogens: an update. *Curr Psychiatry Rep* 5: 347–354.

Halpern, J. and Pope, H.G. (2003) Hallucinogen persisting perception disorder: what do we know after 50 years. *Drug Alcohol Depend* 69: 109–119.

Hermle, L., Fünfgeld, M., Oepen, G., Botsch, H., Borchardt, D., Gouzoulis, E. *et al.* (1992) Mescaline-induced psychopathological, neuropsychological and neurometabolic effects in male volunteers. Experimental psychosis as a tool for psychiatric research. *Biol Psychiatry* 32: 976–991.

Hermle, L., Kovar, K., Hewer, W. and Ruchsow, M. (2008) Halluzinogen-induced psychological disorders. *Fortschr Neuro Psychiat* 76: 334–342.

Lerner, A., Gelkopf, M., Oyffe, I., Finkel, B., Katz, S., Sigal, M. *et al.* (2000) LSD-induced hallucinogen persisting perception disorder treatment with clonidine: an open pilot study. *Int Clin Psychopharmacol* 15: 35–37.

Lerner, A., Gelkopf, M., Skladman, I., Rudinski, D., Nachshon, H. *et al.* (2003) Clonazepam treatment of lysergic acid diethylamide-induced hallucinogen persisting perception disorder with anxiety features. *Int Clin Psychopharmacol* 18: 101–105.

Lerner, A., Oyffe, I., Isaacs, G. and Sigal, M. (1997) Naltrexone treatment of hallucinogen persisting perception disorder. *Am J Psychiatry* 154: 437.

Lerner, A., Shufman, E., Kodesh, A., Kretzmer, G. and Sigal, M. (2002a) LSD-induced hallucinogen persisting perception disorder with depressive features treated with reboxetin: case report. *Isr J Psychiatry Relat Sci* 39: 100–103.

Lerner, A., Shufman, E., Kodesh, A., Rudinski, D., Kretzmer, G. and Sigal, M. (2002b) Risperidone-associated, benign transient visual disturbances in schizophrenic patients with a past history of LSD abuse. *Isr J Psychiatry Relat Sci* 39: 57–60.

Lerner, A., Skladman, I., Kodesh, A., Sigal, M. and Shufman, E. (2001) LSD-induced hallucinogen persisting perception disorder treated with clonazepam: two case reports. *Isr J Psychiatry Relat Sci* 38: 133–136.

Markel, H., Lee, A., Holmes, R. and Domino, E. (1994) LSD flashback syndrome exacerbated by elective serotonin reuptake inhibitor antidepressants in adolescents. *J Pediatr* 125: 817–819.

McGee, R. (1984) Flashbacks and memory phenomena. A comment on 'Flashback phenomena – clinical and diagnostic dilemmas'. *J Nerv Ment Dis* 172: 273–278.

Pechnik, R. and Ungerleider, J. (2004) Hallucinogens. In: Lowinson J., Ruiz P., Milman R. and Langrod J. (eds). *Substance Abuse: A Comprehensive Textbook*, 4th ed. Baltimore, MD: Lippincott Williams & Wilkins, pp. 313–323.

Sierra, M., Phillips, M. and Ivin, G. *et al.* (2003) A placebo-controlled crossover trial of lamotrigine in depersonalization disorder. *J Psychopharmacology* 17: 103–105.

Sierra, M., Phillips, M. and Lambert, M. *et al.* (2001) Lamotrigine in the treatment of depersonalization disorder. *J Clin Psychiatry* 62: 826–827.

Young, C. (1997) Sertraline treatment of hallucinogen persisting perception disorder. *J Clin Psychiatry* 58: 85.