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Flashback Phenomena

Clinical and Diagnostic Dilemmas

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The study of flashback phenomena has been neglected in recent years. A case study and a subsequent literature review examine areas about which conflicting opinions exist. Epidemiological studies have not arrived at universally acceptable classificatory schemes. Clinical approaches vary in accounts of phenomenology of the syndrome and the typology of personalities involved. Etiology remains unknown in spite of numerous theories, most of them not experimentally tested. Diagnostic studies may benefit from recent technological advances such as continuous electroencephalogram, computerized axial tomography scan, cerebral blood flow, and neuropsychological tests. The *Diagnostic and Statistical Manual of Mental Disorders*, 3rd Edition, does not seem to provide an adequate taxonomic niche for this disorder. Therapeutic interventions are examined and their results critically analyzed.

Flashback phenomena are customarily defined as returns of imagery or recurrence of psychedelic drug effects for extended periods after the immediate effect of hallucinogens has worn off. These peremptory and recurrent intrusions of the same—generally frightening—image into awareness occur without volitional control and always after a period of normalcy. They commonly involve the visual sensory system but have been reported in practically all sensory modalities: taste, smell, touch, kinesthetics, vestibular changes, auditory images, as well as distortions of time sense, self-image, or reality sense. They may persist for weeks, months, and rarely for more than a year after the last drug experience (14, 43, 45).

The study of these phenomena has attracted writers, artists, and scientists for decades. While Cooper (6) is credited with the first publication in a medical journal in 1955—a letter to The Lancet, Rosenthal (34) and Horowitz (15), independently, presented the first systematic observations in the English literature. The epidemic of hallucinogen abuse throughout the 1960s gained immediate attention from the media while the medical and psychological literature did not peak until the mid-1970s. In his review of LSD complications, Cohen (5) mentions only Rosenthal's "persistent hallucinosis" which clearly restricts the clinical extent of the observations; in the same paper, in fact, features of chronic anxiety reactions could overlap or be easily ascribed to the flashback experience. Since 1977 very few articles have been published on the subject. Differential diagnostic studies are conspicuously absent, and clinical and experimental substantiation of a variety of proposed etiological theories is also lacking.

We presently report on a recent case of flashback phenomena in which a thorough work-up was done, including the use of the Luria-Nebraska Neuropsychological Test Battery and noninvasive xenon-133 cerebral blood flow studies. The former is a version of A. R. Luria's clinical, diagnostic, and rehabilitative procedures (4, 22) recently standardized by Golden *et al.* (8) at the University of Nebraska. It consists of 269 items designed to sample behaviors across 11 different categories: motoric, rhythmic, tactile, receptive speech, expressive speech, visual, writing, reading, arithmetic, memory, and intelligence. Three additional scales, using items from the above categories, measure right hemisphere function, left hemisphere function, and pathognomonic signs. Cerebral lesion localization profiles for various groups of patients with documented lesion loci have been recently published (21).

The noninvasive xenon-133 inhalation method of measuring regional cerebral blood flow has been well established as a safe and effective procedure (33, 46). This method is completely noninvasive, simultaneously measures cerebral blood flow of both hemispheres, and can be repeated several times in both normal subjects and patients alike. Since regional cerebral blood flow is ultimately controlled by metabolic demands of neuronal activity, it serves as a useful measure of cortical activity and integrity.

We will review the recent literature and discuss the clinical and theoretical implications of the case, with particular emphasis on some of the differences en-

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countered to appropriately labeling this phenomenon in accordance with the newest DSM-III nomenclature.

Case Report

This 19-year-old white, single male, youngest of three siblings, with an eighth grade education and a 5-year history of polydrug abuse, was first admitted in May 1979 because of multiple hallucinatory experiences starting 4 weeks after smoking mushrooms and marijuana. These experiences disappeared initially but then reappeared after smoking marijuana alone, 3 weeks prior to admission. Since then the episodes occurred several times a day, lasted only for a few minutes each time, but are described as "very frightening." In addition to hallucinations, the patient developed numbness in his mouth and lips, as well as tachycardia. He experienced dyspnea and had the sensation of becoming small. There was also a thick, syrupy taste in his mouth and the vision of walls and doors closing in. There was no past history of hallucinations except when taking psylocibine or LSD, roughly 2 years prior to admission.

Past medical history was unremarkable. The physical and routine laboratory work-up were within normal limits. A urine drug screen was negative on admission. Chest and skull x-rays were normal. Brain computerized axial tomography (CAT) scan showed no focal abnormalities, ventricular system was within normal limits, and only an opacification of normally vascularized structures was reported. EEG was not contributory.

Mental status examination on admission showed an alert, oriented for time, place, and person, appropriately dressed young man, appearing his stated age. He was able to carry on a logical, well controlled conversation. Sensorium was intact. Abstraction, judgment, and memory were all intact. There was no evidence of affective swings or suicidal ideation, and no evidence of hallucinations, delusions, or other bizarre behavior. Intellectual assessment with the Weschler Adult Intelligence Scale resulted in Verbal Scale IQ of 88 and a Performance Scale IQ of 94. Bender-Gestalt Test was essentially within normal limits. Rorschach Test and Minnesota Multiphasic Personality Inventory suggested adequate reality contact in a passive, somewhat socially isolated, emotionally immature individual. Tendency to impulsive actions was noted. Finally, it was felt that his experience of flashbacks was "an expression of the anxiety he feels in terms of his relationship with other people and the difficulty he may have in establishing himself in a situation in which he may become independent from his parents."

The patient was observed by the nursing personnel during one of the hallucinatory episodes. Blood pressure and pulse were then within normal limits. From his description and the staff's observations, it was felt that the severe anxiety generated a hyperventilation syndrome which the patient was taught how to control. Throughout this first hospitalization, the patient complained of having flashback experiences on six occasions, almost always described as sensations of "head shrinking, funny taste, numbness, tingling fingers, sweating and the feeling that I'm floating." He was quiet, secluded at times, but mostly pleasant and cooperative. He received no medication and was discharged 14 days after admission to a nearby mental health center. Discharge diagnoses were "drug abuse psychosis" and hyperventilation syndrome.

Almost exactly 1 year later, in June 1980, he was readmitted with a chief complaint of: "I'm losing my mind...I'm about to have a nervous breakdown." While denying any current use of hard drugs, he reported almost daily marijuana usage and occasional drinking. He also indicated that practically since his discharge, and with greater frequency in recent weeks, he was experiencing unusual sensory distortions. He had visual misperceptions regarding size and shape of his own body and the environment around him, as well as seeing objects moving and darting about. The patient became distant and detached from people, and he felt he was retreating into "a different world." Sleeplessness, increased restlessness, and pacing had been predominant patterns at night. His dreams were becoming more and more vivid and threatening to him. His appetite had decreased, with questionable weight loss in the past few months. The sensory distortions were at first happening two to three times monthly for the past year, until roughly 1 month prior to admission. Since then, he felt these experiences daily and almost continuously. The patient was afraid his life was ending. He denied actual auditory and visual hallucinations, but he did have some paranoid ideation (for example, people watching him, his probation officer and counselor plotting to get him back in jail). Fear and anxiety had obviously increased. The patient had considered suicide as an option for relief, but elected to get help before acting on this. He had been on no medications. His physician saw the patient several weeks prior to admission and prescribed oral thioridazine, 10 mg, which the patient took without improvement.

The patient lived with his parents, and maintained a job as an attendant at a school for mentally retarded children. He had a fairly poor job performance in the month prior to admission due to his increasing symptomatology. The patient was concurrently serving 5 years' probation for grand larceny and second-degree burglary. He had served 1 year in jail prior to his first admission. He had been smoking two to three packs

of cigarettes per day for the last 10 years. There was no family history of psychiatric illness.

The physical examination was entirely normal, except for numerous tattoos. The neurological examination was also normal, and the consultant's opinion was that the patient had "probable drug-induced flashbacks with questionable superimposed central lesion." Routine admission blood profile was within normal limits. Basic drug screen on admission and later during the course of hospitalization was negative for phencyclidine, amphetamine, barbiturates, opiates, benzodiazepines, and propoxyphene. Rapid plasma reagin test was negative. An EEG taken the day after admission was reported as "mildly abnormal with suspicious paroxysms of delta activity throughout both hemispheres." Two days later, another EEG showed spike and wave discharge "that appear to emanate from the frontal head region bilaterally and appear to be potentially epileptogenic." A brain CAT scan showed "size of ventricular system in upper limits of normality." Chest and skull x-rays and ophthalmological consultation were all normal.

On mental status examination, the patient showed fair hygiene and good eye contact. His speech showed a normal rate, rhythm, and associations, with no thinking disorder. His behavior was mildly anxious and tremulous. Affect was suspicious, anxious, and upset. He reported visual misperceptions, with micropsia, macropsia, dysmegalopsia, and moving objects. The patient denied auditory hallucinations. Reality-based paranoid ideas were present. There were also some suicidal ideations, but without formed plans or past attempts. Sensorium was intact. Good orientation for time, place, and person; remote, intermediate, and recent memory; and fairly good calculations and attention span were present. Insight was good.

The Minnesota Multiphasic Personality Inventory showed adequate reality contact, social isolation, impulsivity, focus on physical symptoms, rumination, denial, and repression, as well as moderate levels of anxiety and depression.

The patient stayed in the hospital a total of 23 days this time. A trial of phenytoin was initiated, lasted for 10 days, and was discontinued as he showed no improvement. He was later started on a low dose of thioridazine, due to his pervasive anxiety. He responded fairly well to it; the medication was increased up to 50 mg q.h.s. as it improved the patient's sleep pattern and decreased his anxiety throughout the day. In fact he also felt somewhat better about the flashback episodes. The patient entered into group and milieu activities, and benefited somewhat from discussion with peers about his problems, including the heavy drug use in the past. Individual psychotherapy was also used, with several sessions devoted to the fact that the patient would have continuous deficits and that he must try to learn to live with them. Towards the end of his hospitalization he was much more compliant as his general condition seemed to improve. He was discharged home, to the care of his parents, with follow-up at a local mental health center, where he was seen weekly by a mental health worker. He continued to take his discharge medication but still reported flashbacks usually between three and five times a week. Several weeks after discharge from the hospital, the patient was arrested for several traffic violations and was incarcerated in the county jail as his probation from past offenses was violated. The mental health worker has continued frequent contacts with the patient, and reports that, although medication was stopped upon incarceration, the patient reports less frequent flashbacks.

Neuropsychological Evaluation

During the second admission the patient was thoroughly evaluated using the Luria-Nebraska Test Battery. His poorest performance was on the arithmetic scale. The pattern of response indicated, however, that his poor performance was due primarily to a lack of adequate education. The patient's second poorest performance was on the memory scale. His figural memory was found to be relatively intact, while verbal, rhythmic, and kinesthetic memory was somewhat impaired. His memory abilities were further deteriorated by interference-type tasks.

The pattern of deficits suggest significant distractability and dysfunction of a nonfocal quality. Elevations on the intellectual scale were due to a multitude of difficulties. The patient exhibited inadequate education but also a tendency toward concrete thinking and difficulties with visual scanning, concept formation, and ability to abstract. On the tactile scale, he exhibited significant difficulties with fine cutaneous feedback and epicritic (2-point discrimination) touch. These deficits were bilateral and suggestive of parietal lobe dysfunction. He also exhibited mild difficulties with items requiring the expression of relationships between tonal qualities. Finally, his visual scale was mildly elevated due to his poor performance on items tapping visual-spatial and organizational abilities.

The deficits described above are consistent with mild cerebral dysfunction in several areas. There are evidences of frontal, temporal, and parietal dysfunction. Analysis of the Luria-Nebraska lesion localization profile suggested the presence of cerebral dysfunction primarily in both parietal areas and in the right temporal area.

Cerebral Blood Flow Studies

Analysis of the patient's regional cerebral blood flow revealed flow reductions in the left parietal, right

posterior temporal-parietal, and to a lesser extent in the left frontal-temporal areas. These reductions were all approximately 1 SD below the hemispheric mean and are suggestive of reduced cortical activity in these areas.

Discussion

Epidemiological and Clinical Studies

It is interesting that both Stanton and Bardoni's study with 2001 Army personnel entering and leaving South Vietnam (40) and Horowitz's research (14) with 31 representative members of a drug-using community showed a similar prevalence figure (23 per cent) for flashbacks. Matefy *et al.* (25) found that 21 per cent of flashbackers experienced the phenomena daily to few times weekly, 18 per cent two or three times monthly, and 43 per cent once a month to every few months. A significant 64 per cent presented a decreasing frequency of flashbacks after the first experience, but 18 per cent of the patients reported an increase. Holsten (13) found 35 out of 53 flashbackers still troubled by symptoms 1½ to 4 years after the first therapeutic contact. Stanton *et al.* (41) studying 241 respondents concluded that the only drug variable significantly related to LSD flashbacks was marijuana use, which may then be the most reliable predictor of these phenomena, somehow "catalyzing the acid experience both physiologically and psychologically" (p. 67). This was certainly the case with our patient. Marijuana itself has been said to cause flashbacks, but this is considered an extreme version of a more general heightening of the marijuana high that occurs after the use of hallucinogens (10, 25). Matefy *et al.* (25) add alcohol, pleasurable thoughts of situations, and stress to the list of most frequent events triggering flashbacks.

The severity of the flashback phenomena appears to be related to the frequency of hallucinogen use and the amount abused (20, 41). Matefy and Krall (26) found that 57 per cent of the patients described flashbacks as an exact recurrence of the LSD experience whereas 43 per cent said it was similar but not exactly the same. Thirty-five per cent thought the experience was pleasant, but the majority recalled it, as did our patient, as a rather distressing, even frightening, episode. Although more than 75 per cent of flashbackers have a previous direct or indirect knowledge of the phenomenon, 85 per cent of them consider it not or seldom predictable. Finally, from 30 to 50 per cent of people experiencing flashbacks do present, after a careful clinical exploration, additional psychiatric diagnoses, mostly in the realm of personality disorders or underlying psychoses (3, 38). Recently Yago *et al.* (47) have raised the possibility of phencyclidine-induced flashbacks.

Matefy *et al.* (25) list, in order of frequency, perceptual distortions, depersonalization, anxiety, disorientation or confusion, "union with the world," and assorted bodily sensations as the most common clinical characteristics of the flashback experience. Naditch (31) confirms most of these findings. Horowitz groups them as return of perceptual distortions, spontaneous imagery, and recurrent unbidden images. Our patient showed a striking distortion of spatial planes, micropsia and macropsia (dysmegalopsia, according to Jaspers [17]), reflex or synesthetic hallucinations (7), and hyperesthesia. In this context, it is worth mentioning the varieties of flashback phenomena as classified by Houston (16) (sensory, recollective, symbolic, and integral), Juve (18) (recall, hysteria-like, and functional), and Wesson and Smith (45) (perceptual, somatic, and emotional).

Personality studies of flashbackers range from Naditch and Fenwick's (32) findings of "no latent psychopathology" through multiple abnormalities detected in the Minnesota Multiphasic Personality Inventory (38), to the recent case reports of specific obsessive-compulsive (42) and hysterical (35) personality types. Psychological testing has characterized flashbackers diversely as frivolous, spontaneous, outspoken, assertive, active (25), or regressive, immature, maladjusted, vulnerable, passive, and thought-disordered (36). In an experimental study Heaton and Victor (12) concluded that flashbacks "are situationally-induced exacerbations of more pervasive personality characteristics" (p. 89), described as "borderline"-type. Our patient was certainly a passive, immature, dependent, isolated, and anxiety-prone young man, quite ready in a way to fall prey to the flashback experience.

Diagnostic and Therapeutic Issues

The surprisingly high number of etiological theories gleaned from the literature (11, 14, 32, 45) attests to the fact that very little is known about the real causes of the flashback phenomenon. Figure 1 shows the three main categories under which these theories can be grouped. The explanation and critique of each theory goes beyond the scope of this paper. Suffice it only to say that, while it may be didactically convenient to take a multifactorial approach to the disorder, articulating the different components may be operationally difficult and clinically and therapeutically irrelevant. The clinician will in most cases tend to assume that an underlying mental disorder lends some conceptual basis for whatever organic, psychological, or social alteration and/or deficit is taking place. In fact, some of the psychological theories seem to overlap with organic ones. The attention deficit theory is one of the few that has been experimentally tested (24) although with inconclusive results. Judging from the current state of our knowledge, the biochemical

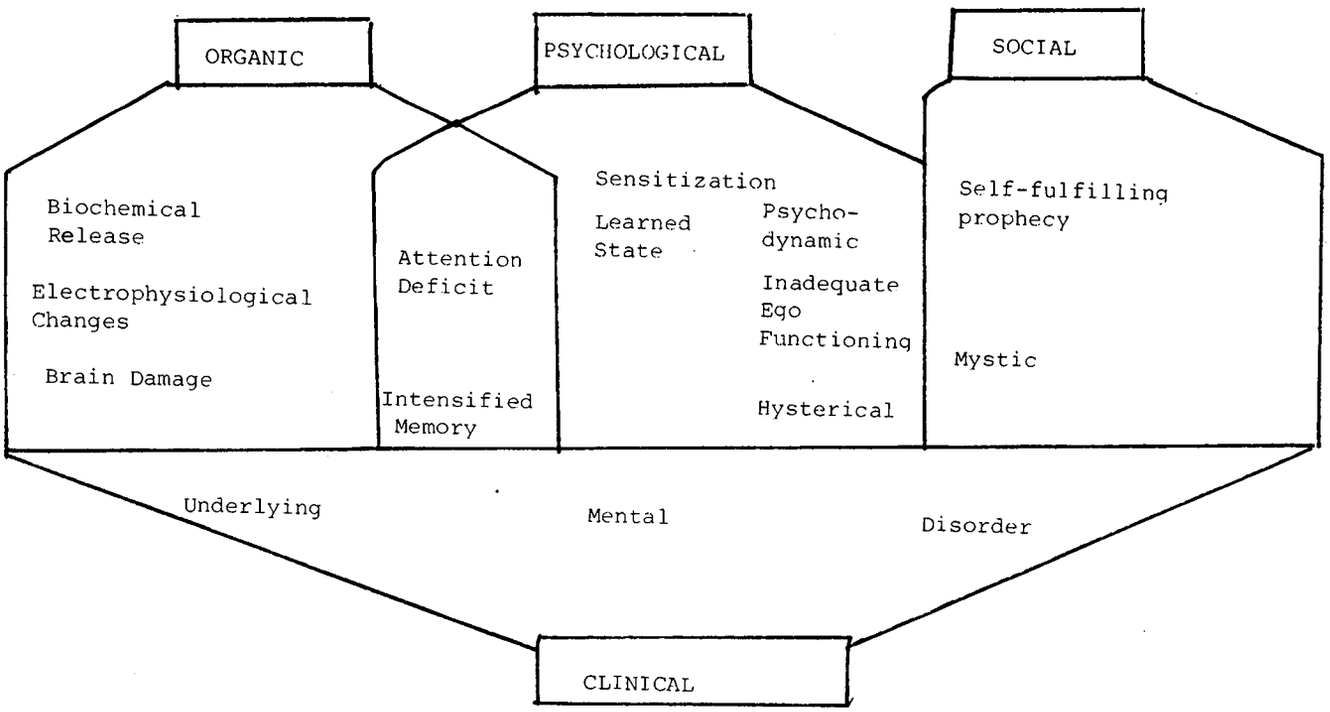

FIG. 1. Flashback phenomena: etiological theories.

release theory (the persistence or building up of the hallucinogen and its metabolites and their release under stressful circumstances) and the sensitization and learned state theories (awareness of changes, experiences recalled when the individual re-enters a state of consciousness similar to the one evoked by the flashback phenomenon) may prove to be the more amenable for research designs.

If the etiology of this clinical phenomenon cannot yet be totally clarified, the mere description of its features has been significantly enhanced by the technological resources now available to the clinicians. The brain CAT scan offers the means for a noninvasive structural study, already used with some advantages in young chronic schizophrenics (44) and older, senile patients (29). To our knowledge it has not been used in a population of flashbackers, and our case presents the ambiguous finding of a picture "in the upper limits of normality." The comparison with the scan done 1 year earlier was equally nonconclusive. However, the potential usefulness of sequential CAT scan studies in patients like this is obvious since they may help to clarify the nature and/or progression of underlying structural brain abnormalities.

Most authors would agree on the consideration of organic factors playing perhaps a decisive role in the production of this symptom complex. In this sense, tests like the Luria-Nebraska Battery and the cerebral blood flow offer helpful ways to better study flashback patients. Previous investigations of neuropsychological deficits in chronic LSD users have suggested significant albeit mild deficits in visual perception, spatial orientation, and abstracting abilities (28). In the present case, difficulties in visual-spatial organization and abstracting abilities were also noted, in addition to deficits in acousticomotor, complex motor, and fine tactile abilities. This could help to explain the complex admixture of clinical symptoms crystallizing in the flashback phenomena. Although the arithmetic scale was the most compromised in our patient's results, it is well known that this scale is extremely sensitive to the proband's educational level and degree of anxiety in the Luria-Nebraska and similar tests. Our findings in any case confirm the existence of a seemingly organic basis for the patient's clinical phenomena.

The same can be said of the cerebral blood flow results. Although the ultimate meaning of flow reductions in several regions of the brain goes beyond the suggestion of reduced cortical activity in such areas (mostly the temporal region), we cannot overlook the striking coincidence between these findings and the EEG findings in our patient's second admission, particularly those referred to the frontal and temporal areas, precisely those involved with complex perceptual cognitive regulatory processes (27). Comparative studies are surely needed.

Clinical phenomena like the flashback syndrome put the American Psychiatric Association's new diagnostic criteria, DSM-III (1), to a test. There could be unanimity in saying that there is hallucinogen abuse (305.3X), and by history and evaluation we could possibly reach consensus on the generic label of this

being a substance-induced organic mental disorder. However, hallucinogen hallucinosis (305.30)—theoretically, the closest diagnostic category—lists as its first diagnostic criterion "recent ingestion of a hallucinogen" (p. 154) which is, by definition, alien to the notion of a flashback phenomenon, unless we stretch the meaning of recent to several weeks or months. Even the term hallucinosis betrays the fact that what the flashbacker experiences is not necessarily hallucinations but illusions or, more appropriately, perceptual distortions. On the other hand, if the observation that marijuana precipitates the flashback experience is correct, one can hardly label it as cannabis intoxication (305.20) since the symptoms of this are entirely different and, again, the time criterion (2 hours) does not necessarily fit with clinical realities. The time lag between actually stopping the hallucinogen intake and the appearance of the symptoms may lead some to think of the flashback syndrome as something akin to a withdrawal or withdrawal-like picture. It is immediately obvious, however, that the flashback syndrome escapes the very basic notion of the withdrawal picture, that symptoms are produced by lack of and urge for the specific substance, and are corrected by the administration of such substance.

Section 2 of DSM-III's organic mental disorder category lists organic hallucinosis (293.82) as a label which could probably apply to the flashback phenomenon. Unfortunately, the issue is muddled by what seems to be a contradiction between the explanatory note of section 2 ("...etiology noted from outside the mental disorders section of ICD-9-CM or [is] unknown" [p. 162]) and criterion C of each and every diagnostic category under the heading: "Evidence...of a specific organic factor...etiologically related to the disturbance" (p. 116). At best, this label would negate even the consideration of a hallucinogen as the possible cause of the flashback phenomena. Of course, it can be argued that the DSM-III book makes the proviso (p. 101) that "under certain circumstances a reasonable inference of an organic factor can be made from the clinical features alone," but then the whole purpose of a thorough diagnostic approach can lose its meaning. Thus it seems that DSM-III cannot provide a safe diagnostic niche for this peculiar entity.

Are the flashback phenomena but one variety of temporal lobe epilepsy? Some EEG findings and the clinical symptomatology (7, 37), the perceptual disorders in particular, might suggest the likelihood of this diagnosis. In addition, experimental studies and therapeutic reports (35, 41) seem to demonstrate that hallucinatory flashback phenomena may represent seizure activity. Arguments to refute this hypothesis are not few, however: EEG patterns are not always specific; symptoms in most cases do not have the sudden onset or termination characteristics of temporal lobe discharges; some cases do not respond to anticonvulsants and on the contrary do respond to phenothiazines, (19) known in turn to lower the seizure threshold (9). This was, in fact, the case with our patient. An intriguing finding, however, was that his EEG was much worse on the second admission. We cannot assert the reasons for this other than speculating about the eventual influence of a continuous and obviously higher marijuana abuse throughout the intervening year on the neurophysiological structures. This would indirectly mean that drug abuse causes actual functional impairment of the CNS. The debate about these issues will no doubt continue, and systematic inquiries with continuous EEG, neuropsychological testing, cerebral blood flow studies, and long term treatment with anticonvulsants, carbamazepine in particular (42), seem to be promising approaches.

Treatment of this condition is mainly oriented to alleviating the patient's mounting anxiety, correcting patterns of behavioral response to the perceptual phenomena, and attempting to eliminate the actual symptoms, in this order. Psychotherapeutic approaches include primarily brief, supportive, reality-oriented techniques aimed at reassuring the patient of the short duration and non-life-threatening nature of the symptoms (18); behavioral techniques including relaxation, activity-oriented, and desensitization techniques (23) have been used. Still, in some cases, psychodynamically oriented psychotherapy (35) may be useful at disentangling the symbolic or the interpersonal meaning of some of the perceptual distortions experienced by the patient. On the other hand, pharmacotherapy of the flashback phenomena has usually meant the more or less empirical use of phenothiazines (2), butyrophenones (30), benzodiazepines (39), vitamins (45), and as mentioned above, anticonvulsants. An objective assessment of the response to these diverse agents is made difficult by the self-limited course of the syndrome and the lack of adequate follow-up studies. Studies like that of Moskowitz (30) lack in control group and objective measurement of changes. Thurlow and Girvin's report (14), although anecdotal, appears consistent and significant. Our finding was that the patient did not respond to phenytoin and seemed to do better with thioridazine, but certainly we cannot conclude much from this observation.

Thus, the flashback phenomena remains a fascinating and elusive subject, and many questions are still unanswered. Why do not all hallucinogen users experience flashbacks? Is the organic etiology necessary and sufficient? Does the nature and extent of hallucinogen use predispose to the production of flashbacks

through causation of some sort of anatomical or functional lesion? What is the most appropriate diagnostic label for this syndrome and where does it fit in the current diagnostic terminology? Is there a specific treatment approach? Neither our case study nor the preceding review, of course, answer these questions. Our patient may have had the detected organic deficits long before the flashback experience came to the fore; or our findings may be somewhere specifically related to the nature of the CNS changes induced by the chronic hallucinogen use and, therefore, to the experiencing of the flashback phenomena. More systematic studies along those lines are certainly needed.

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