Hallucinogens: a Cause of Convulsive Ergot Psychoses

Sylvia Dahl Winters

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ABSTRACT

HALUCINOGENS: A CAUSE OF CONVULSIVE ERGOT PSYCHOSES

By

Sylvia Dahl Winters

Ergotism with vasoconstriction and gangrene has been reported through the centuries. Less well publicized are the cases of psychoses associated with convulsive ergotism. Lysergic acid amide, a powerful hallucinogen having one-tenth the hallucinogenic activity of LSD-25 is produced by natural sources. This article attempts to show that convulsive ergot psychoses are mixed psychoses caused by lysergic acid amide or similar hallucinogens combined with nervous system lesions secondary to vasoconstrictive effects of ergotoxine.
HALLUCINOGENS: A CAUSE OF CONVULSIVE ERGOT PSYCHOSES

By

Sylvia Dahl Winters

A Thesis in Partial Fulfillment of the Requirements for
the Degree Master of Science in the Field of Psychiatry

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Each person whose signature appears below certifies that this thesis in his opinion is adequate, in scope and quality, as a thesis for the degree Master of Science.

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Throughout history epidemics of chronic ergotism have been reported. Galen (c130-c200 AD) (1) described infection of standing grain in the field. Galen denoted such fungi by the term erysibe. Robert (2) considered that erysibe denoted the poisonous ergot fungi Claviceps. However, Lieber (3) states that Robert, an experienced Russian pharmacologist identified all cereal borne disease as ergotism. An epidemic is described in France in 994 AD as "apparently a hidden fire burnt up their limbs in a single night and caused them to drop off" (4). This epidemic reportedly caused 40000 deaths (5). Under the terms ignis sacer, mal ardens, and St. Anthony's Fire are described illnesses associated with famine or unwholesome cereals probably representing ergot poisoning. Caporael (6) strongly suggests that convulsive ergotism was the physiological basis for the Salem witchcraft trials in 1692. The afflicted persons were often stricken with violent fits and had hallucinogenic symptoms and perceptual disturbances accompanying ergotism. Even in the nineteenth century, belief in witchcraft was still prevalent and many believed the sufferers from convulsive ergotism to be possessed by demons.

The last extensive German epidemic of convulsive ergotism occurred near Frankenburg in upper Hessen in 1879-1881. Physicians calculated there were five hundred cases with five percent mortality. This epidemic is important because over sixty patients with psychoses were
observed over a period of twenty years by neurologists and psychiatrists (Siemens, Tuczek, and Jahrmaker).

Barger (7) distinguished two distinct types of ergot poisoning: convulsive east of the Rhine and gangrenous west of the Rhine. Severe mental derangements were limited to the convulsive type. Ergotoxine is the cause of gangrenous ergotism. Barger suggested a factor in addition to ergotoxine as the cause of convulsive ergotism. In the convulsive form there were many intermediate stages from mere formication to epileptiform convulsions. Barger described spasms beginning in the fingers and as the patient would walk around the toes also became affected. Spasms might begin in the toes and extend upward. In more severe cases generalized convulsions occurred. These paroxysms lasted a few minutes to several hours and were extremely painful. Frequently cases differed from true epilepsy in that patients remained conscious. In severe cases the tongue was lacerated and occasionally bitten off. In the gangrenous form symptoms were mild unless the critical limit was exceeded and gangrene set in.

CONVULSIVE ERGOT PSYCHOSES

Tuczek (8,9) reported hospitalized cases from the psychiatric clinic of Marburg. These were cases of convulsive ergot psychoses from the Frankenburg region.

Case 1. Previously healthy seven year old girl admitted June 1881 after ergot poisoning winter 1879. Her mother and uncle died from ergot poisoning and other family members became ill. At admission
she talked endlessly about imaginary ideas, ran about, made faces, laughed, cried, and hit people. She had absent knee reflexes but otherwise normal physical exam. There were no epileptic attacks but repeated bedwetting was noted. She was discharged to home September 1881. Follow-up at age twelve showed low intelligence but otherwise mentally intact while absent knee reflexes continue. She does not remember being hospitalized.

Case 2. Twelve year old boy. At admission March 1881 the following noted: psychological alienation, fearful, confused, increased motor movements, talked incessantly about imaginary things, laughter inappropriate, poor control of urination, absent knee reflexes but otherwise normal physical exam. A few days after admission noted to occasionally repeat a single sentence a hundred times. He had confused memories of school and home. He cleared and was discharged home August 1881. Readmitted October 1881; had cramp attacks, periods of coma, confusion, and epileptic attacks. His attacks increased, some with loss of consciousness. He died April 1884.

Case 3. This twenty one year old male had ergot poisoning Fall 1879. He lay senseless in bed between epileptic cramp attacks during December 1879. At admission June 1880 he had absent knee reflexes but otherwise normal physical exam. In July 1880 he had generalized convulsions, bit tongue, with loss of consciousness. At discharge July 1881 low intelligence noted but otherwise normal mental functioning. At follow-up May 1886; no further decrease of intelligence noted, no
cramps, no seizures, absent knee reflexes continue and he is employed by a farmer.

Siemens (10) felt the nervous system symptoms were secondary to pathological poisons in the nervous system. Jahrmarker (11) evaluated Frankenburger patients twenty years after the ergot epidemic. Some were recovered, some continued to have epileptic attacks, some were weak minded and some had continued to have recurrence of disease without further ergot ingestion.

Reformatzky (12) reports on the 1889 Russian ergot epidemic in Gouvernement Wiatka.

Six types of psychological sickness from ergot:

1) weak minded, lack interest in surroundings, inert, apathetic, disoriented, sleepy; memory, comprehension and ability to relate facts noticeably weakened; speech unclear, slow; sometimes nightmares, delusions, and epileptic attacks take place.

2) weak mindedness without lack of interest in surroundings. Not as disoriented, moods changing, sometimes elated, sometimes depressed.

3) acute weakness of mind with hallucinations, fear, excitement.

4) hallucinatory disturbances

5) negative fixed idea; melancholy, fear, depression

6) different levels of mental disturbances

Neurological findings: Altered pain, tactile and temperature sensation. Pupil reflexes sometimes sluggish. Normal or absent knee
reflex. Romberg sometimes positive. Painful cramps, generalized cramp attack without loss of consciousness, tonic cramps of single muscle groups, epilepsy, long lasting nervous system damage. Some recover in a few months with others the sickness is long lasting.

In 1908 Glushkoff (13) described a case of acute ergot psychosis. The psychological sickness was characterized by the author as a rapid disturbance of ability to associate ideas and the harming of the intellectual processes. Epilepsy and somatic symptoms occurred. This case ended with recovery.

Gurewitsch (14) at the psychiatric hospital Buraschewo (Russia) reports eighteen case histories with psychological symptoms from the 1909 ergot epidemic in Twerschen Gouvernement. The poor and ignorant suffered the most.

Case 1. This thirty-seven year old male sickened suddenly fourteen days prior to admission. He sprang up, danced, prayed, cried, and could not sleep. His speech was meaningless and he did not understand why. He had the idea that the lamp was some type of a treasure and burned his hand. The scars were still visible at admission. He was very fearful especially when he saw water and he admitted he had poor memory for the events of his illness. During admission he was quiet, calm, depressed and disoriented. He had no neurological abnormalities or epilepsy. He was hospitalized for two months. At times he was disoriented and confused. On the third day of hospitalization he
suddenly sprang to the window, beat himself, spoke incoherently, was soon calm, then amnesic for the event. At discharge he had no psychological or neurological abnormalities.

Case 2. About eleven days prior to admission both patient and his wife became ill. He was restless, fearful, and had tingling in his extremities. He had crazy ideas and delusions and began to see all types of things at night but was unable to remember them. At admission concentration poor, restless, fearful, clouded consciousness, painful cramps arms and legs but no neurological abnormalities. During hospitalization; fear, depression, poor comprehension, but no hallucinations observed. He had poor memory for events of the illness and discharged healthy one month later.

Case 3. This is the twenty-seven year old wife of Case 2. She heard voices, noises, gunshots, and thought her husband was the devil so she bit and scratched him. At time of hospitalization, she was agitated, disoriented, poorly coherent, and had cramps in arms and legs with normal neurological exam. Three days later awareness of surroundings cleared and she did not remember how she got to the hospital. During rest of hospitalization she had clear sensorium, normal intellect, seldom had fearful feelings and was discharged healthy five weeks later.

Von Bechterew (15) reports a host of neurophysiological disturbances with ergot poisoning which he called psychoses of self. The first psychological symptoms included apathy and feeling of ants crawl-
ling inside. Many became ill on the same day if large amount of ergotized rye were eaten, or sometimes up to three weeks later. The most distressing physical symptoms are cramps in the limbs similar to those in epilepsy. Cramps are also in facial, abdominal, back or any other muscles of the body. If the muscle cramps are in their feet or hands they cannot move them. Any attempt to stretch the cramped area causes the cramp to become stronger and more painful. With time these cramp attacks may result in loss of consciousness. The cramp attacks especially in the extremities normally occur for many weeks or months. After an attack the patient falls asleep and is later exhausted. They have dizziness, visual disturbances, loss of consciousness, short intense sight and auditory hallucinations, they act as if they were in their hallucinations, and illusions play a noteworthy role. After several attacks many patients become mentally disturbed in a stuporous condition lasting a few hours or weeks.

Ergot sickness is usually chronic. Many patients after they have seemingly recovered experience flashbacks of the illness. Repeated ergot ingestion was ruled out as the cause of the recurrence of the illness. The cause of the reappearance of the acute form seems to be cooling of the body, strong emotional moods, and poor nutrition. After having seemingly recovered many patients still complain of physiological weakness, weakness of memory and being too lazy to think. Psychopathic heredity apparently does not play a role in the appearance of this disease.
LYSERGIC ACID HALLUCINOGENS PRODUCED IN NATURE

Barger reported that a large number of grasses are infested with some twenty species of *Claviceps*. *Claviceps purpurea* frequently infects rye, while wheat, barley, and oats are rarely infected (7). The *Claviceps* fungus parasitizes the developing ovaries of the rye producing the long sclerotia which replace individual grains on the rye plant. These sclerotia produce numerous ergot alkaloids. The alkaloid content varies with the locality, soil, climate conditions, species and strain. Wet spring and summer weather favors ergot infestation of the rye.

The natural alkaloids isolated from ergot include the ergotamine, ergotoxine, and ergobasine groups (16). More than forty individual alkaloids have been isolated from *Claviceps*. Upon hydrolysis these natural alkaloids yield lysergic acid or the amide, lysergic acid amide. Floss, Tcheng-Lin, Kobel and Stadler (17) report *Claviceps purpurea* produces ergocornine and ergokryptine, which are both ergotoxine group alkaloids. Corbett, Dickerson, and Mantle (18) report *Claviceps purpurea* produces principally the ergotoxine group with minor components of ergotamine and traces of ergotaminine and ergotinone. Castagnoli and Mantle (19) report the isolation of D-lysergic acid in cultures of *Claviceps purpurea*. They state this is the first identification of D-lysergic acid in pure cultures of *Claviceps purpurea*. Arcamone, Bonino, Chain, Ferretti, Pennella, Tonolo and Vero (20) report the isolation of a hallucinogen, lysergic acid amide from the Stevens and Hall strain of *Claviceps paspali* after rye embryo propagation. This lysergic
acid hallucinogen has also been isolated from morning glory seeds. Hofmann and Tschetter (21) isolated lysergic acid amide from Mexican Rivea corymbosa seeds and seeds of Ipomea tricolor. Taber, Vining and Heacock (22) also isolated lysergic acid amide from the seed of several varieties of Morning Glory (Ipomoea and Convolvulus species). D-lysergic acid amide is a powerful hallucinogen having one-tenth the activity of LSD-25. D-lysergic acid amide occurs in natural sources and is a probable cause of convulsive ergot psychoses. There are several other closely related lysergic acid hallucinogens which have been synthesized and possibly some of these hallucinogens also occur naturally and are also a cause of convulsive ergot psychoses. The hallucinogenic effects of lysergic acid diethylamide were discovered by Hofmann in 1943 when he inadvertently ingested a small quantity of LSD-25. Rothlin (23) reports active hallucinogen LSD-25 being eight to ten times as potent as monoethylamide of D-lysergic acid (LAE). Hoffer and Osmond (5) report the psychological effect in man of several lysergic acid derivatives synthesized and examined by Sandoz Laboratories with hallucinogenic ratings as follows:

D-lysergic acid diethylamide (LSD-25)-100
D-lysergic acid amide-10
D-lysergic acid ethylamide (LAE-32)-5
D-lysergic acid dimethylamide (DAM-57)-10
Interestingly Persyko (24) reports LSD type of psychosis after ingesting methysergide (Sansert) a semi-synthetic ergot alkaloid. Reportedly sixteen mg of Sansert is equivalent to 100 mcg of LSD in "street use" (25). It seems possible that the concentrations naturally occurring are so low that the other lysergic acid alkaloids mask these and the hallucinogens are not detected in the routine ergot assay. Either lysergic acide amide or one of several closely related LSD-like hallucinogens occurs in the ergotized rye and is therefore a highly probable cause of the convulsive ergot psychoses.

LSD PSYCHOSIS

LSD-25 (26)
Somatic symptoms: Nausea, dizziness, loss of appetite, blurred vision, paresthesia, weakness, drowsiness, trembling, frequently associated with sympathomimetic effects (increased pulse, slight increased temperature, mydriasis, piloerection). Psychological symptoms: delayed forty to sixty minutes and most intense after one to two hours. The predominant mood change is euphoria but may quickly be followed by profound depression and anxiety. There may be unmotivated giggling or laughter with tears. One may have a sense of well being or oppressive malaise with depersonalization and disturbances of perception. Visual hyperaesthesia is common with visual experiences of unparalleled purity, intensity, brilliance and novelty. Commonly illusions and occasionally hallucinations.
Dewhurst and Hatrick (27) identified differentiating features of LSD Psychosis. Suggestive symptoms include loss of time sense, grandiose delusions of a pseudo-philosophical nature together with visual hallucinations, perceptual disturbances and commonly regression to childhood. Perhaps the most striking clinical feature, however, is the wide variety of the schizophreniform, affective and psychoneurotic symptoms present in the same patient. Spontaneous recurrences are complications of LSD psychosis for which there is no adequate explanation. Some experience recurrence of frightening delusions or hallucinations after an interval of normality ranging from days to months after their last ingestion of LSD. They report nineteen cases requiring three to sixteen weeks of hospitalization and chemotherapy.

Cohen and Ditman (28) state "illusinogen" would be a more appropriate term than hallucinogen for almost invariable distortions of perception elaborated from sensory cues. They described several prolonged reactions to LSD.

Case 1. Thirty six year old legal secretary became schizophrenic ten days after an LSD treatment. She required several hospitalizations over a two year period.

Case 2. Thirty two year old secretary took LSD two hundred to three hundred times over a three year period. She had also taken other hallucinogens. Frightening, spontaneous recurrences of the LSD hallucinatory phenomena were almost daily events.
Case 3. Forty one year old chronically depressed office manager received LSD eight times and remained psychotic for two years.

Case 4. This ten year old boy ingested 100 mcg LSD and remained abnormal for one month.

Case 5. Male hypnotist received twenty five sessions of LSD therapy and remained psychotic for seven months.

Case 6. Psychoanalyst took 100 mcg of LSD which resulted in hypochondriacal agitated depression for eight months with slow but complete recovery.

DISCUSSION

In most cases LSD psychosis is of limited duration. However, there are many reports of prolonged psychosis after LSD ingestion. The reported cases of convulsive ergot psychoses probably represent a prolonged LSD type of psychosis in combination with nervous system lesions. Obviously there are neurological findings (absent knee reflex, epileptiform cramp attacks, generalized seizures and actual central nervous system and spinal cord lesions in severe cases) which cannot be explained by the known effects of LSD alone.

The foregoing review of data and literature shows the following symptoms occurring both in LSD psychosis and convulsive ergot psychoses: perceptual distortions or illusions, fearfulness or panic, excitement, confusion, misinterpretations, delusions, mood changes and, perhaps, especially significant, flashbacks. It is also relevant that these
phenomena occur abruptly in previously healthy individuals who generally return to a nonpsychotic state after the toxic effects have worn off. The similarities and differences are listed in the following table.

Table 1—Comparisons Between Convulsive Ergot Psychoses and LSD Psychosis (Immediate and Prolonged)

<table>
<thead>
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<th>Similarities</th>
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<tbody>
<tr>
<td>perceptual distortions or illusions</td>
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<td>fearfulness or panic</td>
</tr>
<tr>
<td>excitement</td>
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<tr>
<td>mood changes</td>
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<tr>
<td>depression</td>
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<tr>
<td>confusion</td>
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<td>misinterpretations</td>
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<tr>
<td>delusions</td>
</tr>
<tr>
<td>flashbacks</td>
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<tr>
<td>some cases of lengthy duration</td>
</tr>
<tr>
<td>occurred in previously healthy individuals after ingestion of toxin</td>
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<tr>
<td>subject generally returns to nonpsychotic state</td>
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<table>
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<tr>
<th>Differences</th>
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<tbody>
<tr>
<td>The following are found in Convulsive Ergot Psychoses but not in LSD Psychosis</td>
</tr>
<tr>
<td>muscle cramp attacks</td>
</tr>
<tr>
<td>frequently absent knee reflex</td>
</tr>
<tr>
<td>occasionally Romberg positive</td>
</tr>
<tr>
<td>occasionally epilepsy</td>
</tr>
<tr>
<td>occasionally central nervous system lesions</td>
</tr>
<tr>
<td>occasionally spinal cord lesions</td>
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A review of the literature shows that lysergic acid amide, an LSD type hallucinogen has been produced by *Claviceps paspali* in culture after propagation on rye embryo. To date this hallucinogen has not been identified in naturally occurring ergotized rye or the bread baked from this rye. To make the evidence conclusive the ergot produced by various *Claviceps* should be analyzed for lysergic acid amide and other lysergic acid hallucinogens. If any further cases of convulsive ergot psychoses occur then an attempt should be made to isolate the toxic material ingested and assay for hallucinogens. The serious neurological consequences of ergot ingestion prohibit intentional human ingestion.

The evidence shows a powerful hallucinogen, lysergic acid amide, is produced by *Claviceps* which infests rye. This article has shown that convulsive ergot psychoses which occurred after ingestion of rye bread containing ergot produce a clinical picture compatible with prolonged LSD psychosis. The evidence, therefore, points toward convulsive ergot psychoses as a mixed syndrome; an LSD type of psychosis combined with nervous system lesions.
REFERENCES


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