CASE REPORT

Joshua A. Perper,[1] *M.D., LL.B., M.Sc.*

Fatal Strychnine Poisoning—A Case Report and Review of the Literature

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ABSTRACT: A typical case of suicidal strychnine poisoning by a rodenticide is presented. The forceful muscular convulsions were accompanied by a clear sensorium. Pathological findings consisted of an early onset of postmortem rigidity and microscopic hemorrhages with minimal degenerative neuronal changes in the spinal cord. The highest tissue concentrations of strychnine were found in the bile and liver. The pathophysiology and epidemiology of strychnine poisoning is reviewed and discussed in context.

KEYWORDS: pathology and biology, suicide, poisons, strychnine

The strychnine poisoning fatality presented in this report represents the first such occurrence in the past 20 years at the Coroner's Office of Allegheny County (Pittsburgh), during which more than 20 000 autopsies were performed, all of which were screened toxicologically.

Recent national statistics also indicate the rarity of strychnine poisoning. The most recent statistics released by the National Center for Health Statistics indicate that, in 1981, 13 deaths occurred which were the result of strychnine poisoning [*1*]. This is consistent with the 1978 total of 13 [*2*]. The recognition of strychnine intoxication is, however, important because prompt treatment has been reported to be lifesaving, even when the victim was exposed to a very large dose of poison.

The presented case is particularly instructive because of the typical clinical manifestations, the spinal cord pathology, and the toxicological findings.

Case History (A84-513)

In the noon hours of a May day, loud moans of pain were heard from within a locked apartment of a modern complex building located in a middle-class neighborhood in a suburb of Pittsburgh. The disturbance prompted the neighbors to call the police and emergency medical personnel, who eventually forced entry into the apartment at about 12:49 p.m.

The tenant, a 56-year-old man, was found lying on the floor in the living room, face down, and moaning. He was, however, fully conscious and related that, at about noon, he had, with

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[1]Coroner, Allegheny County Coroner's Office, Pittsburgh, PA.

suicidal intent, ingested half a can of mole poison. A 140-g (5-oz.) can of "Mole-Nots" rodenticide, still containing 64 g of green pellets, was found on a table in the same room. The label of the can indicated that it originally contained 140 g with a 0.35% concentration of strychnine sulfate (Fig. 1).

The patient, who was moved promptly to a couch, complained of severe abdominal pains. He was then observed to have an approximately 30-s attack of tonic convulsions, with extreme body rigidity and opisthotonus, followed by a period of total flaccidity. During the tonic phase of the attack, the patient "turned blue" and stopped breathing. After an additional attack, which occurred seconds later, he became unconscious and collapsed before the emergency medical team had a chance to take his vital signs. Cardiopulmonary resuscitation was initiated and the patient was ventilated, but to no avail. The electrocardiogram showed several ventricular complexes over a 10- to 20-min period without any perceptible pulse.

Autopsy Findings

The autopsy was performed 3 h after death. The body was that of a well-developed, well-nourished man, weighing 79 kg (175 lbs) and measuring 173 cm (68 in.) in length. Though the body was warm, and showed only slight postmortem lividity, it was extremely rigid.

Pertinent findings included the presence of 61 g of green, thick, mortar-like material in the stomach, of a similar color as the contents of the poison can. The mucosa of the stomach was unremarkable (Fig. 2).

The brain weighed 1100 g and was unremarkable. In particular, no edema was evident. The upper cervical cord appeared congested. The lungs were congested and edematous. Incidental findings included chronic emphysema of the lungs and slight arteriosclerosis of the aorta.

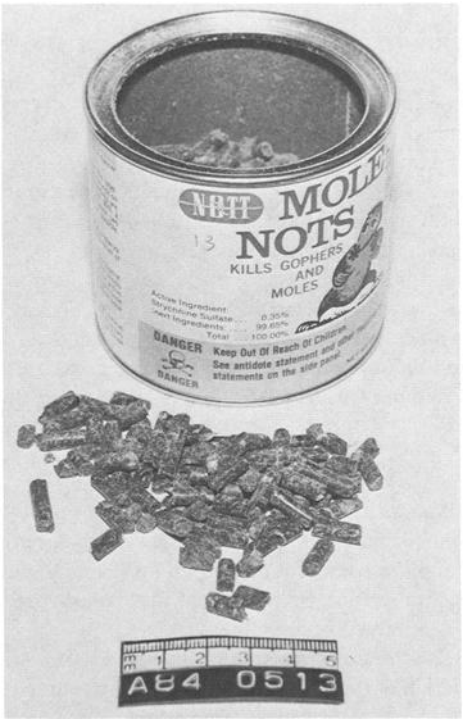

FIG. 1—*Can of "Mole-Nots."*

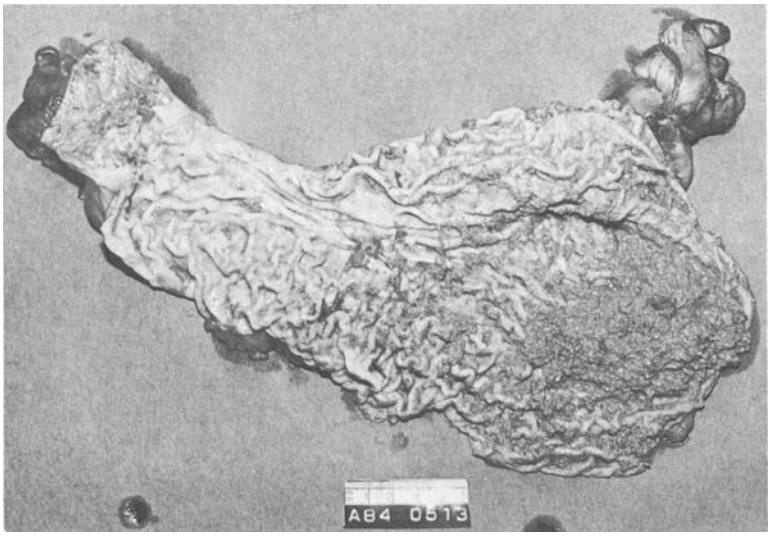

FIG. 2—*Stomach.*

The light microscopic examination of the spinal cord revealed in both anterior and posterior horns, though mainly in the former, multiple petechial hemorrhages. Ring hemorrhages were seen around the capillaries. Some of the neurons showed chromatolysis (Fig. 3). An electron microscopy examination of multiple spinal cord sections did not reveal any additional findings.

Toxicology Findings

Samples of blood, bile, vitreous fluid, and urine, as well as various body tissues, were analyzed for strychnine content with gas chromatographic procedures, with quinine added as an interval standard to the sampled aliquots. The samples were also analyzed for the presence of drugs, including alcohol, carbon monoxide, cyanide, barbiturates, benzodiazepam, and methaqualone. A general screen for acid, basic, and neutral drugs was also performed. No other drug was identified in the body besides strychnine (Table 1). The total amount of strychnine found in the stomach was 213.5 mg.

Besides the stomach contents (17.5 mg/L), the highest concentrations of the poison were found in the stomach wall (1.49 mg/L), bile (0.92 mg/L), and liver (0.62 mg/L). The concentrations in the blood and kidneys were 0.33 and 0.32 mg/L, respectively. There was either no strychnine present or amounts too small to detect in the vitreous fluid, muscle, and brain.

Discussion

Strychnine is an alkaloid which was first isolated in 1818 from St. Ignatius beans (*Strychnos ignatii*), a woody vine native to the Phillipines. The commercial source of the drug is, however, the dried, ripe seed of another species of the Strychnos plant, *S. nux-vomica*, a vine of India. *S. nux vomica* usually contains from 1.1 to 1.4% of strychnine, together with an equal amount of a related, toxic alkaloid named brucine.

Both strychnine and its salts are crystals which dissolve in either water or alcohol, and have a bitter taste. The elucidation of the chemical structure of strychnine (Fig. 4) in 1921 by Robert Robinson and Robert Burns Woodwars was hailed, at the time, as a great achievement of analytical chemistry.

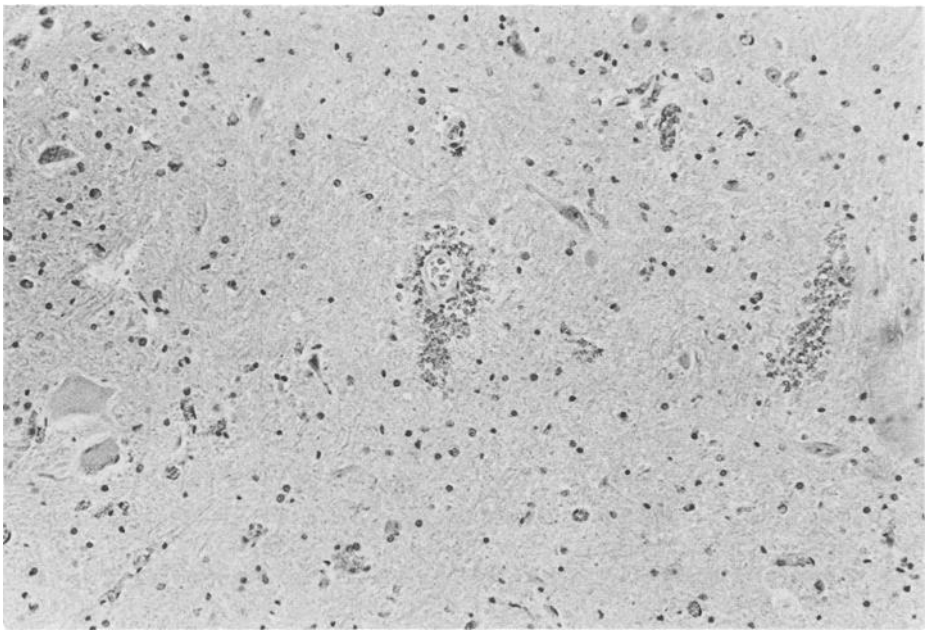

FIG. 3—*Some of the neurons showing chromatolysis.*

TABLE 1—*Concentration of strychnine in tissues and body fluids.*

Source	Concentration, mg/L
Stomach contents	175.0
Stomach	14.9
Bile	9.2
Liver	6.2
Small intestines	4.1
Blood	3.3
Kidney	3.2
Plasma	2.6
Urine	1.4
CSF	0.08
Muscle	too low to quantitate
Cerebrum (occip.)	too low to quantitate
Midbrain	not detected
Medulla	not detected
Pons	not detected
Cerebellum	not detected
Vitreous fluid	not detected
Colon	not detected

As late as 1965, strychnine was used quite extensively in the American pharmacopeia, mainly as a tonic and a cathartic, usually in conjunction with aloin, belladonna, cascara, and podophyllin, in amounts varying from 0.5 to 1.3 mg per tablet. Its great toxicity, coupled with the lack of clinical and experimental proof of its therapeutic efficacy, led to its elimination from the current pharmacopeia. The *American Drug Index*, 1984 [*3*], still lists strychnine as available in combination with yohimbine HCl and methyltestosterone (as an aphrodisiac [sic])

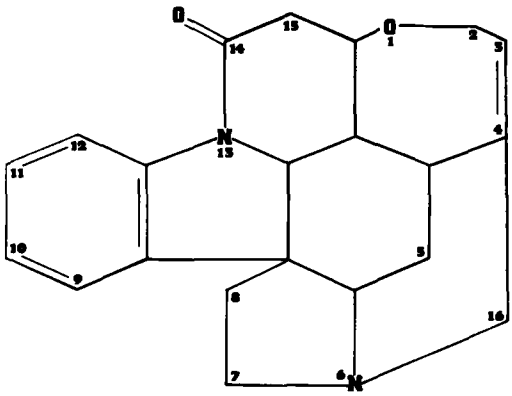

FIG. 4—*The elucidation of the chemical structure of strychnine.*

and in combination with a thyroid extract. However, current medications containing strychnine are no longer found in American pharmacies.

The early medicinal use of strychnine led to a significant number of accidental poisonings, mainly in children who were attracted by the chocolate or sugar coating of the strychnine-based tablets [*4–5*].

Currently, the major remaining sources of strychnine poisoning are rodenticides and pesticides. However, strychnine is used in the illicit manufacturing of narcotics (for example, cocaine), and a number of accidental strychnine poisonings have been reported in drug addicts, who mistakenly sniffed the white, powdery, and bitter strychnine in the belief that it was cocaine [*6, 7*].

In the past century and in the first quarter of this century, strychnine was apparently quite a favorite of poisoners. During the prohibition years, some criminals developed the unsavory habit of offering cocktails of strychnine in whiskey to undesirable associates and then following them afterward to watch their ghastly agony and death [*8*].

In recent years, the most common form of strychnine poisoning is suicidal ingestion of liquid or solid rodenticides or insecticides.

An unusual, intentional, though not suicidal, self-administration of strychnine has occasionally been practiced by members of the Free Pentecostal Holiness Church, a southern religious sect living in Kentucky, Tennessee, Virginia, and North Carolina. Some members of the group, known as "saints," have exposed themselves to poisonous snakes or open fires and have knowingly ingested strychnine sulfate as a proof or divine test of their faith [*9*].

Clinical Manifestations

The symptomatology of strychnine poisoning is very dramatic and, as in the case reported here, is characterized by very powerful tonic contractions that are easily triggered by the slightest noise. Horizontal pendular nystagmus is also reported [*10*].

The differential diagnosis includes other convulsive conditions, such as epilepsy, tetanus, meningitis, and phenothiazine overdose. The fact that the person is often conscious until seconds before death, in spite of severe acute poisoning, distinguishes strychninization from the various forms of epilepsy and convulsions caused by other toxic substances. The lack of a prodrome, the absence of fever, and the sudden onset of convulsions exclude meningitis and meningoencephalitis. The relatively clear sensorium, coupled with the powerful and painful convulsions, arouses panic in the patient. The tonic and tetanic contractions of the diaphragm and thoracic and abdominal muscles stop the respiration and cause cyanosis and marked

anoxia. The convulsions usually occur at intervals of 10 to 15 min, and may result in death. Prompt treatment usually results in uncomplicated recovery.

Cases have been reported, however, in which the survivors of an intoxication have developed complications related to the strong muscular contractions, such as profound lactic acidosis, hyperthermia, and rhabdomyolysis [*7*] or myocardial damage as a result of anoxia [*11*].

Mechanism of Poisoning

Strychnine-induced convulsions are not due to a direct excitatory effect, but to a decrease in neuronal inhibition. Strychnine blocks the uptake of the inhibitory neurotransmitter glycine at the postsynaptic receptor site in the motor neurons of the ventral horn of the spinal cord (Fig. 5). Attempts to use this property of strychnine as a rationale in the treatment of glycinosis, a glycine storage disease, have yet to be proven successful.

Pathological Findings

The marked postmortem rigidity observed in the presented case, in spite of the short postmortem interval, is easily explained on the basis of the preceding violent muscular contractions.

Strenuous antemortem activity hastens the appearance of postmortem rigor as a result of intramuscular lactic acidosis.

The literature reports scanty or nonspecific pathological changes. The author could not find any reference to petechial or ring hemorrhages in the spinal cord, though neuronal degenerative changes of the neurons have been reported in experimental animals [*12*]. The spinal cord hemorrhages may be nonspecific and related either to anoxia or mechanical injury caused by violent convulsions, but the absence of similar findings in other tissue locations make these explanations less likely.

Toxicology

The usual lethal dose of strychnine is reported to be between 50 and 100 mg; however, there are reported cases, especially in children, of lethal doses between 15 and 30 mg, and even as

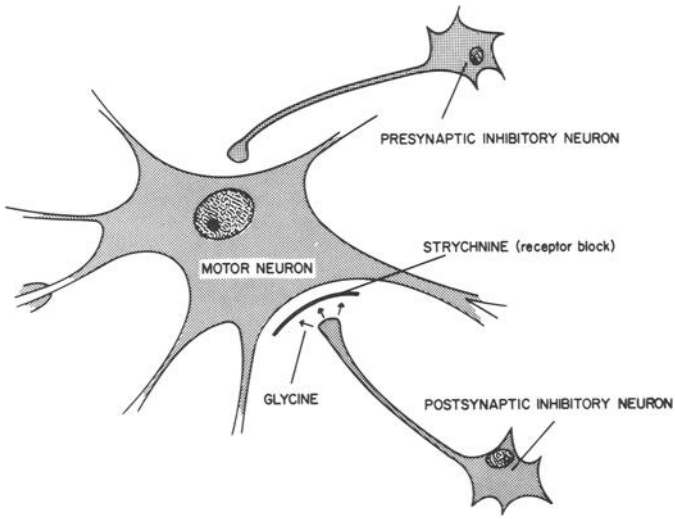

FIG. 5—*Illustration showing how strychnine blocks the uptake of the inhibitory neurotransmitter glycine at the postsynaptic receptor site in the motor neurons of the neural horn of the spinal cord.*

low as 5 to 10 mg [*13*]. On the other hand, people have survived doses several times larger than the usual lethal dose, as high as 3750 mg [*11,7*].

The principal organ of detoxification has been reported to be the liver, which clears close to 5/6 of the ingested dose. The liver apparently stores the poison temporarily, because there are reports of delayed, recurrent intoxication because of a secondary release of the strychnine stored in the liver. The kidneys excrete 10 to 20% of the dose.

Surprisingly enough, there are very few reports in the literature on the differential concentrations of strychnine in various tissues in humans [*14*]. Our data support the fact that the liver is the major detoxifier, as its differential strychnine concentration is two to three times that of other organs.

The results also indicate that strychnine is concentrated and excreted in the bile, which shows the highest concentration of the drug. The reported secondary strychnine related symptoms are probably due to a delayed emptying of the gallbladder.

Treatment

The treatment of strychnine poisoning consists of prompt control of convulsions with intravenously administered diazepam or short-acting barbiturates [*15,16*]. Usually 10 mg, intravenously, of diazepam are sufficient to control the convulsions [*6*]. Muscle relaxants such as succinylcholine or *d*-tubocurarine and mechanical assistance of respiration may also be required [*13*]. Gastric lavage is recommended following, rather than preceding, the control of convulsions. Obviously, in the presented case, the death occurred too soon after the discovery of the poisoning to permit effective treatment without the full resources of a hospital resuscitation system.

Summary

A typical case of suicidal strychnine poisoning by a rodenticide is presented. The forceful muscular convulsions were accompanied by a clear sensorium. Pathological findings consisted of an early onset of postmortem rigidity and microscopic hemorrhages, with minimal degenerative neuronal changes in the spinal cord. The highest tissue concentrations of strychnine were found in the bile and liver. The pathophysiology and epidemiology of strychnine poisoning are reviewed and discussed in context.

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Address requests for reprints or additional information to
Joshua A. Perper, M.D.
Allegheny County Coroner's Office
106 Crofton Dr.
Pittsburgh, PA 15238