A CASE OF POISONING DUE TO EATING POISON-HEMLOCK (CICUTA MACULATA)

WITH A REVIEW OF REPORTED CASES *

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The plant discussed in this paper, Cicuta maculata or, as it is more commonly called, water-hemlock or poison-hemlock, is a widely distributed plant in this country and has for centuries been known as a plant possessing a deadly poison. A number of cases of death have been reported by American and European physicians, but the cases undoubtedly are more numerous than those reported, as it is not always possible to ascertain the history of a case, a considerable number of the victims being children. The European form of the plant is called Cicuta virosa. It is found well distributed over the whole of Europe, but seems to vary somewhat in the activity of its poison in different localities.

It was not until the year 1876 that an approximate knowledge was obtained of the active agent in this plant. Boehm¹ published the results of a careful investigation into the characteristics of the poison and its action on certain animals and came to the conclusion that it was a resin. This was fatal to cats in doses of 5 cg. per kilo weight when given through the mouth and in doses of 7 mg. when given intravenously. The fatal dose for frogs was 2 to 3 mg. This substance Boehm has given the name of cicutoxin and regards it as the active agent of Cicuta virosa. alkaloid coniin is also believed to be present. The poisons are found in all parts of the plant but chiefly in the root. When tried on animals it was found to be slowly absorbed through the intestinal tract; the effects at times would not appear until after the lapse of several hours. If vomiting took place it would very often save the life of the animal. Boehm was unable to find the exact place of absorption. When it was injected intravenously the action was very rapid, but absorption through the skin was slow. One observer states that it is popular knowledge in some parts of Europe that if slices of the roots of Cicuta virosa are placed on the backs of frogs convulsions will set in.

^{*}This is not the poison-hemlock of historical interest. It is supposed that the cup of poison-hemlock drunk by Socrates was the *Conium maculatum*, a plant that has been introduced into this country.

^{1.} Boehm: Arch. f. exper. Path. u. Pharmakol., 1876, v, 279.

The history of the case seen is characteristic of poisoning due to eating Cicuta maculata. The mother of the patient gave me the following details:

History.—The patient's family history was negative. She had always been well. There was no history of convulsions. While the patient was working with her father and younger brother in the field they found some strange-looking roots, which she tasted, and as they had a rather pleasant taste she chewed and swallowed some. (The exact quantity eaten could not be ascertained.) A few minutes afterward she fell over unconscious and had what her father called an attack of cramps. From this time on she was entirely unconscious and had convulsions every ten or fifteen minutes. She vomited several times and in the vomitus could be seen particles of the swallowed root. She was at once taken to the home.

Examination.—I first saw the patient about three quarters of an hour after the onset. She was entirely unconscious and slightly cyanotic; the eyes were dilated; the pupils did not react to light; there was loud, heavy breathing through nose and open mouth. Examination of mouth showed considerable amount of mucus and saliva, but was otherwise negative. The thorax was well formed. Fine râles were heard throughout both lungs. The heart was negative, 85 beats to minute. The pulse was of fair quality and force, normal in rate and rhythm, 85 to minute. The abdomen was negative.

Course of Illness.—Just after the completion of the examination the patient had one of her convulsive attacks of which three were observed. These began with twitching of the muscles around the mouth and eyelids, spreading rapidly to the muscles of the chest, arms, abdomen and lower extremities, until finally the muscles of the whole body were firm, hard and rigid, the forearms and hands slightly flexed and fingers clenched. The legs were extended and slightly parted; the head was slightly raised from the pillow, but the shoulders and buttocks rested on the bed. At the climax of the tonic convulsion the muscles of respiration appeared to be fixed, the face becoming markedly livid and cyanotic. Finally, when the patient appeared to be on the verge of suffocation, clonic convulsions would set in, the muscles would relax, and gradually the body would again come to rest. At the height of the attack the heart-beat would rise to 125-130 per minute.

The patient had no movements of the bowels or bladder during an attack, but froth and blood from a slight wound in the tongue appeared at the mouth.

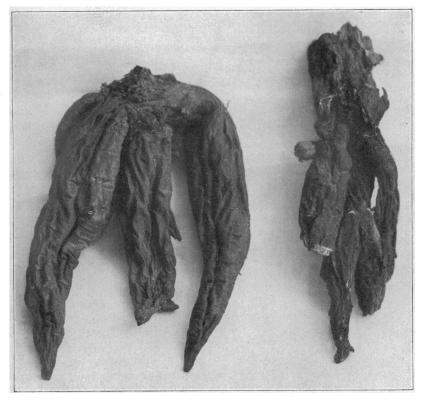
These frightful convulsions were very different from any other convulsions that might be thought of under the circumstances, and when the mother showed me the root from which the child had eaten, it was decided at once to wash out the stomach. Dr. Heising was called in and with his assistance the stomach was emptied, though with the greatest difficulty on account of the violent convulsions.

At once the convulsions stopped, but a period of extraordinary restlessness appeared, lasting for about three hours, during which the patient tossed about aimlessly in bed from side to side and from one end to the other, striking her head against the bed or wall unless prevented from so doing. She was given doses of bromids and gradually became quiet; she remembered nothing, however, from the time the root was eaten at noon on Monday until the succeeding Wednesday evening. During this period she was very constipated and the bowels moved only by enemas. The stools presented nothing of particular interest. The bladder was unaffected. After the third day the patient made an uneventful recovery. The only thing worth mentioning was a stiffness and soreness of the whole body due, no doubt, to the convulsions.

It was learned that the father and the younger brother, a boy of 7, working with the patient had also tasted the roots. The father had chewed a bit of the root and spat it out, but shortly afterward felt dizzy

and sick at the stomach. This passed over in a short time. The boy, however, shortly after eating a small quantity, became unconscious, had one convulsion and vomited the swallowed material. When seen he was conscious and resting quietly in bed but felt sick at the stomach. Ife made an uneventful recovery.

I sent the root shown me to Professor Ravenel, of the State Hygienic Laboratory at Madison, Wis. He kindly reported to me the results of an examination made by Dr. Dennison, of the department of botany of the



Partially dried roots of Cicuta maculata.

State University, who found that the root belonged to a plant popularly known as American water-hemlock, musquash-root, beaver-poison, children's bane, death of man, musquash-poison, poison-hemlock, poison snakeweed, spotted parsley, wild parsnip and wild hemlock, and botanically known as *Cicuta maculata*.

In the available literature forty-six cases of poisoning were found reported by European and American authors. The European physicians speak of the plant as *Cicuta virosa*; Professor Abel informs me, however,

that those who have studied the two plants believe that they are very similar and possibly are identical. Certainly the clinical histories show no differences in symptomatology.

In looking over these histories the first point to be noticed is the great number of children among the victims, thirty-two out of a total of forty-six. This is to be expected, as few adult persons would eat the root except by mistake. A considerable number of the adult victims mistook the roots for spikenard, carrots, and a number of other edible roots. One person was given the roots cooked in a vegetable soup with homicidal intent. One other person had the root served with cooked vegetables with no knowledge of its toxic properties. The results seemed to be just as violent when the roots were eaten after cooking as when they are taken uncooked. Chevallier² reports five most interesting cases. A family of five was troubled with severe itching; for relief the skin was rubbed with Cicuta aquatica, with the result that all developed toxic symptoms and two of the children died. It has already been mentioned that it is possible to produce convulsions in frogs by rubbing the skin with slices of Cicuta virosa.

The symptoms of onset were very much the same in all the cases. Pain and discomfort in the region of the stomach were the first symptoms mentioned in five cases. This came on shortly after eating and varied in severity from a discomfort to severe pain; in one case the pain became a general distress.

The most characteristic and pronounced symptoms, however, were nausea, vomiting, and convulsions. These appeared in a few cases while the patients were still eating the roots; but in most of the cases from half an hour to two hours afterward. In several reports mention is made of the fact that the vomitus contained particles of the eaten root. The convulsions in typical cases are most violent and many of the observers speak of them as epileptiform. Bloody froth at the mouth is a usual occurrence, but no mention is made of involuntary movement of bowels or bladder. One adult person was killed by a fall sustained while in convulsions. A fatal termination occurred in twenty-one cases.

Of the minor symptoms may be mentioned:

Dizziness is spoken ofin	4 cases
Buzzing in earsin	2 cases
Arhythmia of heartin	3 cases
Violent beating of heartin	3 cases
Small and slow pulsein	6 cases
Pulse fast (120-140 per minute)in	1 case
Pulse moderate (90 per minute)in	2 cases
Weak respirationin	1 case
Uneven respirationin	3 cases
Rapid respiration (40-45 per minute)in	2 cases

^{2.} Chevallier: Jour. de chim. méd., 1836, series 2, ii, 606.

Paralysis and numbness of limbsin	2	cases
Stuporin	2	cases
Twitching of musclesin	1	case
Dryness of throatin	1	case
Feeble palate reflexin	1	case
Conjunctiva bloodshotin	1	case
Swollen abdomenin	1	case
Abdomen not rigidin	1	case
Pain in region of heartin	1	case
Feverin		case
Dilated pupilsin	21	cases
Facial pallor and cold skinin	11	cases
Unconsciousnessin		cases
General weaknessin	2	cases
Anxious expression of facein	1	case
Skin warm and dryin	1	case
Very mobile eyeballsin	1	case
Trismusin	1	case
Hallucinationsin	l	case
Deafness and confusion, etcin	1	case
Flushed facein	1	case
Pupils respond to lightin	1	case
Pupils did not respond to lightin	1	case
Diarrheain	2	cases
Sweatingin	3	${\it cases}$
No sweatingin	1	case
Polyuriain	1	case
Double visionin	1	case

From this it will be seen that we have here a root containing a poison capable of producing a great variety of symptoms of varying degrees of importance. It is noteworthy that the observation made by Boehm experimentally that in cases in which early and free vomiting took place, often the life of the animal would be saved, is borne out clinically. Early and free vomiting is a most favorable occurrence and in the great majority of patients recovering, this took place either spontaneously or through the administration of emetics. There is some evidence to show that the vomiting is due to an action on a center in the brain.

Autopsies are reported in only five cases. The only findings worth mentioning are slight redness and injection of the gastric and upper intestinal mucosa and the presence of the eaten roots as the exciting agent. No change of particular significance can be pointed out. In one case a spotted condition of the spleen is mentioned.

The poisonous properties of the root are not lost by drying; but Boehm states that exposure to a temperature of 100 C. always diminishes its activity. Clinically, however, as has already been noted, cooked roots have produced death with all the important symptoms caused by eating the uncooked root. The ethereal or alcoholic solution of the poison may retain its toxic properties for years when kept at an ordinary temperature.

Boehm believes that the active principle acts on a center in the medulla oblongata, causing convulsions, stoppage of respiration, increase in blood pressure and vagus irritation. Experimentally cicutoxin has been found to produce in frogs: (1) violent clonic and tonic convulsions; (2) acceleration of respiration, which, however, ceases during the convulsive seizures; (3) slowing of the heart and, during the convulsive seizures, a long diastolic pause. All of these effects Boehm ascribed to an action on Heubel's center for convulsions, supposed to be located at the point of the calamus scriptorius.

The experimental effects on mammals may be summarized as follows: diarrhea, salivation, muscular twitchings, increased frequency of respiration appear shortly after administration of the drug; then there sets in violent convulsions affecting all the voluntary muscles of the body. During these convulsive attacks there has been noted a stoppage of respiration but a marked increase in the force of the heart-beats. Usually the contents of the urinary bladder are forced out, possibly owing to the violent contractions of the abdominal muscles. The convulsions last from about half a minute to two minutes. In the intervals the respirations are deep; the pupils are not always dilated but just before death dilatation appears. Death occurs usually at the height of a convulsive attack. Experimentally no characteristic local effects have been produced. Bennevitz³ noted congestion of the blood-vessels of the brain in guinea-pigs. This, he concluded, might be due to a narcotic action of the poison.

The mode of action, and the toxic and pharmacological properties of the active agent or agents in this plant need to be studied over again.

Other poisons of the same group to which cicutoxin belongs are picrotoxin, obtained from Anamirta paniculata, cenanthotoxin, the active principle of Enanthe crocata; coriamyrtin, which occurs in several species of Coriaria; phytolaccotoxin, prepared from a Japanese species of Phytolacca, which may possibly also be obtained from Phytolacca decandra or pokeberry. A number of the digitalis series contain bodies which may produce the same symptoms as cicutoxin and the other members of the picrotoxin group. Among these are toxiresin, obtained from digitoxin, digitaliresin, from digitalin, and oleandresin, from oleandrin. These bodies all produce powerful stimulation of the central nervous system, more especially of the areas around the medulla oblongata. Two alkaloids, samandarin and samandaridin, obtained from the skin of the newt, appear to resemble the members of the picrotoxin group in their effect.

Cushny⁴ states that picrotoxin and its allies act chiefly on the medulla oblongata, while the spinal cord and the higher parts are little affected. He believes that there is no necessity for believing in the existence of a convulsion center, as intense stimulation of the medulla will produce clonic contractions of the muscles throughout the body. The medulla is

^{3.} Bennevitz: Med. Ztg., 1836, v, 51.

^{4.} Cushny: Pharmacology and Therapeutics, 1901, p. 425.

not, however, the exclusive seat of action for in many animals the reflexes are found to be increased when the medulla is severed from the cord and this indicates that the spinal cord is also more excitable than normally. Cushny states, in his text-book on pharmacology, that the action of the picrotoxin series on the spinal cord is best seen in the fish and reptile, but in the higher animals the action is more confined to the region of the medulla oblongata.

An interesting observation has been made by Christison,⁵ in Scotland. He found that during the month of August the roots, leaves and fruit were almost without effect. This, Huseman⁶ believes, is not due to climate, as in those parts of Scandinavia with almost the same climate the *Cicuta virosa* is decidedly poisonous during that month, so there must be other conditions affecting its activity.

Very meager data can be obtained as to the quantity of the root capable of producing death in an adult person. Prof. Charles Lee⁷ states that a dram and a half has been known to destroy human life, but in view of Christison's observation just mentioned it may be that the activity of the poison varies with the seasons and with the locality. Lieutenant Carpenter,⁸ U. S. A., states that two men who had eaten a whole root died in an hour and a half; a third who had eaten only a very small quantity recovered.

In the diagnosis of a case the following are the important points to be kept in mind, as shown in the case-histories quoted and the case of the patient observed.

- 1. The eating of parts of a strange plant and especially of the root, resembling that of the poison-hemlock.
- 2. The characteristic symptoms, pain in the region of the stomach, nausea, vomiting, and the frightful convulsions appearing usually within an hour. Especially significant is the presence of parts of the eaten plant in the vomitus. Unconsciousness is a condition seen in all of the cases of which I have had an oral description—four in number. In the cases reported in print, however, it is mentioned in only nine cases. Final proof is, of course, furnished by the demonstration of parts of the plant.

With a history of eating of a wild plant, with pain and discomfort in the region of the stomach, nausea and vomiting, and convulsions supervening within an hour or one hour and a half, the presumption is justifiable if the case is seen in this country, that the plant eaten is *Cicuta maculata*. Other poisonous wild plants found in the United States that are capable of causing convulsions are: black cherry (*Prunus serotina*);

^{5.} Christison, quoted by Pribram: Arch. f. Krim.-Anthrop. u. Kriminalist, Leipsic, 1900, iv, 166.

^{6.} Huseman, quoted by Pribram: Arch. f. Krim.-Anthrop. u. Kriminalist, Leipsic, 1900, iv, 166.

^{7.} Lee, Charles: quoted in Tr. Minnesota State Med. Soc., 1871, Vol. I, 53-55.

Jamestown or jimson weed (Datura tatula and Datura stramonium); black nightshade (Solanum nigrum); and the sneeze-weed (Helenium autumnale). The convulsions produced by these plants are not, however, so severe as those produced by poison-hemlock.

In regard to the treatment there is not much to be said. Meyer used a decoction of gallnuts in one case with very satisfactory results. Wine of antimony (tartar emetic) and zinc sulphate has been used in several, causing free emesis. The stomach-tube is, however, the most satisfactory on account of the quick and sure results obtained. It may also be well later to wash out the bowel thoroughly and give an active cathartic. The ordinary methods for the comfort of the patient, suitable bed-clothing, hot-water bottles to extremities where indicated, sponge baths, and bromids if the patient is very restless, are simple, efficient methods of treatment that have been used in many cases with good results. To control the very severe convulsions before the removal of the poison, chloroform and chloral have been suggested.

Considerable damage is done every year to cattle, which eat the tubers, or may be poisoned in marshes by drinking water contaminated by the juice of the roots, which may have been crushed by being trampled on. Melted lard is recommended for the treatment of cases of poisoning among cattle.

In regard to prophylaxis two thoughts will at once come to mind: (1) the education of the public to recognize our poisonous plants; and (2) the extermination of poisonous plants. Both of these methods can be employed, and as far as the Cicuta maculata is concerned the eradication of this plant in well-populated portions of the country would not be a difficult undertaking, if the people were taught to recognize it and to know its poisonous properties. In these days of preventive medicine, instruction in the recognition of our principal poisonous plants, about thirty in number, could be given in the departments of botany in our high schools, colleges, universities, and agricultural schools, and would undoubtedly result in the saving of a considerable number of human lives annually, as well as in the prevention of suffering experienced by thousands of people every year.

Note.—In addition to the authorities cited in the text, the following may be found of interest:

Caillard: Clin. d. hôp., Paris, 1829, iv, 33.

Chesnut: Principal Poisonous Plants of the United States, Bull. 20, U. S. Dept. Agri., Washington, D. C.

Hazeltine: New England Jour. Med. and Surg., 1818, vii, 219.

Henning: Mitt. a. d. Geb. d. Med., Altona, 1836, iv, 84.

Hwass: Upsala Lakaref. Förh., 1877-8, xii, 262.

Jawandt: Jour. d. pract. Arznk. u. Wundarznk., Jena, 1798, v, 588.

Kelp: Vrtljschr. f. gerichtl. Med., 1879, new series, xxx, 380.

^{9.} Meyer: Med. Ztg., 1842, xi, 178.

Little: Clinic, Cincinnati, 1874, vii, 49.

Maly: Oesterr. med. Wchnschr., 1844, pp. 1065, 1097.

Matchett: Cincinnati Lancet and Obs., 1870, new series, xii, 462. Stockbridge: New England Jour. Med. and Surg., 1814, iii, 334.

Trousdale: Brit.-Am. Jour., 1862, iii, 37. Wilson: Lancet, London, 1871, ii, 396.

Folk: Tr. South Carolina Med. Assn., 1882, xxxii, 69.

Mossberg: Eira, Stockholm, 1889, xiii, 435.

Pohl: Arch. f. exper. Path. u. Pharmakol., 1894, xxxiv, 259.

Pribram: Arch. f. Krim.-Anthrop. u. Kriminalist, Leipsic, 1900, iv, 166.

Boehm: Arch. f. exper. Path. u. Pharmakol, 1874-5, iii, 216.