BOTULISM

A FURTHER REPORT OF CASES OCCURRING IN THE PACIFIC COAST STATES *

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In a previous report¹ it was shown that prior to September, 1917, there had been twenty-two recorded outbreaks of botulism in the United States, in which eighty-one persons were poisoned and fifty-five died. In this collection of cases one important outbreak had been overlooked. In January, 1912, Dr. A. R. McCracken² of Seattle reported a series of six cases of poisoning by home-canned asparagus, in which three of the patients died. In all, therefore, there have been at least twenty-three recorded outbreaks of botulism in this country, of which nineteen occurred in the Pacific Coast States. The mortality of these recorded cases was 66.6 per cent.

In addition to the outbreaks in which human beings were affected there were also at least six instances in which domestic fowl were poisoned and showed symptoms which were identical with those produced experimentally by feeding with the toxin of *Bacillus botulinus*.

ETIOLOGIC FACTORS

It is an important fact that, of the twenty-five instances of poisoning of human beings or domestic fowl in which the source of the poisoning was determined, seventeen were shown to have been caused by the ingestion of home-canned vegetables or fruits, and in three instances it was established that the poisoning was due to the toxin of *B. botulinus*, by the recovery of *B. botulinus* from portions of the discarded food.

Since September, 1917, there have been at least ten more outbreaks of botulism affecting human beings and four affecting domestic fowl or animals, and of these fourteen outbreaks, thirteen have been shown to be due to the ingestion of home-canned vegetables or fruits. Seven of the outbreaks affecting human beings and all of those affecting domestic animals and fowl occurred in the Pacific Coast States, and

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1. Dickson, E. C.: Botulism: The Danger of Poisoning from Vegetables Canned by the Cold-Pack Method, Jour. Am. Med. Assn., 1917, **69**, 966.

2. McCracken, A. R.: Botulism, Leucocyte, 1912, **19**, 80.

from remnants of the home-canned products which caused three of them, *B. botulinus* was recovered. One of the remaining outbreaks occurred in Fallon, Nev., one in Decatur, Ind., and one in Newark, N. J. I have been informed that *B. botulinus* was recovered from the discarded food in two of these. It is probable that the complete report of these outbreaks will be recorded by the physicians who cared for the patients.

DANGER IN HOME CANNING OF PERISHABLE FOODS

The increase in the number of outbreaks of botulism in this country bears a very interesting relationship to the marked increase in the practice of home-canning of perishable foods which has taken place during the past few years. The home-canning of vegetables is a development of comparatively recent years, but on account of the increased cost of food products since the beginning of the war, and especially since the inauguration of the program for the conservation of foods during the past year, the amount of vegetables which are canned at home has been tremendously increased. Coincidentally with the increase in the amount of home-canning of vegetables as well as of fruits there has been a marked increase in the number of outbreaks of botulism in the United States, and it is extremely interesting that not only is there an increase in the number of instances of poisoning of human beings, but there are also more frequent occurrences of poisoning of domestic fowl and animals by feeding spoiled home-canned products (Table 1).

TABLE 1.—SHOWING THE OCCURRENCE OF RECORDED OUTBREAKS OF BOTULISM DURING THE PAST NINETEEN YEARS, AND THE IMPORTANCE OF HOME-CANNED PRODUCTS AS THE CAUSE OF THE POISONING

Period From To	Interval	Number of Outbreaks		Source of Poisoning			
				Home-canned		Food of Animal Origin	Unknown
		Human	Domes- tic*	Vegetable	Fruit		
1899 - 1903	5 years	2		...	...	2	...
1904 - 1908	5 years	1	Fowl 1	1	...	1	...
1909 - 1913	5 years	10		5†	1	3	1
1914 - Sept. 1917	3¾ years	10	Fowl 5	10	1	1	3
Oct. 1917 - Mar. 1918	½ year	10	Fowl 3 Hog 1	10	3	...	1

* The outbreaks listed under "Domestic" were instances in which only fowl or animals were affected and do not include those in which human beings and fowl or animals were coincidentally poisoned.

† One outbreak was caused by commercially-canned vegetables.

A. REPORT OF OUTBREAKS IN WHICH HUMAN BEINGS WERE POISONED *

OUTBREAK 8 (three cases).—On Sunday, Nov. 25, 1917, Mrs. E. of Seattle, opened a jar of home-canned asparagus and cooked half of the contents. None of the persons who ate the cooked asparagus suffered any ill effects. On the following evening she "warmed up" the remainder of the asparagus from the jar, by placing it for a few minutes in warm but not boiling water. Her husband stated that this asparagus did not taste very good, but he ate it all. On Tuesday afternoon Mr. E. complained of disturbance of vision, was nauseated and vomited. On Wednesday morning he was very weak, vomited again after taking food, and had severe diarrhea. The diarrhea continued during the day and following night, and during the night the patient complained of cramps in the legs. There was no abdominal pain during this time, and no disturbance of sensation. During Wednesday afternoon he began to have difficulty in talking. On Thursday Mr. E. was unable to sit up because of weakness, and he complained that he could not hold up his head. He was unable to speak intelligibly, and he complained of dryness in the mouth and pharynx. During the afternoon he began to have difficulty in swallowing, and by evening "all the water returned through his nose." He had much difficulty in clearing thick, tenacious mucus from the pharynx and had severe strangling spells when he attempted to swallow. There was no disturbance of mentality, no pain except the cramps in the legs, and no fever. He was found dead in bed early Friday morning, about two hours after he had succeeded in swallowing a small amount of milk.

On Thursday, November 29, another jar of the same lot of asparagus was opened and was served cold as salad at the Thanksgiving dinner. Two persons partook of the salad and both developed symptoms of poisoning and died. One patient was seen by Dr. O. F. Lamson, who placed his records at my disposal. The clinical course of Dr. Lamson's case was practically identical with that of the case which has been described, although there was much more severe strangling when the patient attempted to swallow. The temperature varied from 97 to 98.8 F., and the pulse from 92 to 128 per minute. The patient died on the fourth day after eating the asparagus salad.

Through the courtesy of Dr. Lamson and Coroner Tiffin of Seattle, I was permitted to do partial necropsy on the bodies of two of the victims, although a coroner's necropsy had been previously performed in each case, and both bodies had been embalmed. There was moderate congestion of the meninges and of the abdominal and thoracic viscera in each case, and apparent bronchopneumonia in one. No macroscopic hemorrhages were seen. On microscopic examination a few typical thrombi were found in each case, in one occurring in the larger meningeal arteries, and in the other, occurring in the lungs. No disintegration of the Nissl granules was detected.

The remnants of the salad which had been served on Thanksgiving day were fed to the chickens, and all the chickens developed typical symptoms of limber-neck, and died. Bacteriologic examination was made of the contents of the crops and gizzards of ten of the chickens, and from six of them a virulent strain of *B. botulinus* was isolated. This strain is labeled Strain 7 of my series.

The asparagus was canned according to the directions published on page 19 of the pamphlet issued by the Kerr Glass Manufacturing Company, with the exception that it was not parboiled or blanched before it was packed in the

* In detailing the case histories only those facts are described which are essential for establishing the diagnosis. For a more complete discussion of symptomatology and pathology refer to Dickson, E. C.: Botulism: A Clinical and Experimental Study. Monograph of the Rockefeller Institute for Medical Research, No. 8, 1918, in which Outbreaks 1 to 7 of my series are described.

jars. The material was purchased in the city market, washed in cold water, packed into 1-pint and 1-quart Mason jars which had been boiled, and covered with cold water to completely fill the jars. One-half teaspoonful of salt was added to each jar and the covers were loosely applied. The jars were immersed to the neck in a wash-boiler which had a tightly fitted cover, and were allowed to remain for three hours after the water began to boil actively. On removal from the boiler, the jars were tightly sealed and placed in a dark closet.

OUTBREAK 9 (one case).—On Wednesday, Dec. 5, 1917, Miss B. of Ontario, Ore., opened a jar of home-canned string beans and tasted the contents to determine whether they were good. She decided that they were "not exactly right" and discarded them. On the following day she complained of blurred and double vision and difficulty in articulation, and consulted Dr. Rollo A. Payne of Ontario, Ore., who sent me the records of the case.

When Dr. Payne first saw the patient there was bilateral blepharoptosis, dilatation of the pupils and difficulty in articulation. There had been no nausea or vomiting, no pain and no diarrhea. On the following day the blepharoptosis was more marked, speech was unintelligible and the patient was very restless, being compelled to struggle for breath. There was great difficulty in swallowing. Examination of the urine showed nothing abnormal, and the blood pressure was 142 mm. (systolic). Miss B became rapidly worse and died about fifty-two hours after tasting the beans. Dr. Payne stated that the symptoms were those of a bulbar paralysis which "interfered more with the respiratory and throat muscles than with other groups." Death was apparently from respiratory failure.

The discarded beans were thrown to the chickens, and thirty-nine of them died of limber-neck within two or three days after eating them.

It was not possible to learn the details of the method which was followed in canning the beans.

OUTBREAK 10 (two cases).—On Friday, Jan. 11, 1918, Dr. P. M. Savage of San Bernardino, Calif., was called to see Mrs. J. who gave the following history:

On Thursday afternoon she had noticed that she "saw double," but was otherwise quite well. On Friday morning she was very weak, and was scarcely able to walk; in fact, she had fallen three times while preparing breakfast. Just before Dr. Savage was called, the patient had staggered to a neighbor's house for help, but collapsed before reaching the door. When first seen she was seated in a chair, without apparent pain, and apparently without any disturbance of mentality. She complained of weakness, double vision and photophobia. There was bilateral blepharoptosis, dilatation of the pupils and loss of reflex to accommodation, though not to light. Articulation was very deliberate and indistinct. When the patient stood she became dizzy and there was incoordination of muscular movements. The skeletal reflexes were intact and there was no appreciable disturbance of sensation. The temperature was 99.5 F. and the pulse rate was 100.

On Saturday morning there was still photophobia and double vision, and the patient complained of great fatigue. There was difficulty in swallowing and the patient had severe strangling spells when she attempted to swallow. During the morning articulation was distinct, although husky, but by evening speech was unintelligible. During the day she became rapidly worse, swallowing became more difficult and strangling spells more frequent. There was much distress from the accumulation of thick, tenacious mucus in the pharynx. There was persistent constipation, but urination was not disturbed. The temperature varied from 97.8 to 99.5 F., and the pulse rate from 80 to 100. The patient died early Sunday morning.

Meantime on Thursday night, Miss H., a blind woman to whom Mrs. J. was companion, developed severe cramps in the abdomen with nausea, vomiting and diarrhea. She became extremely nervous, almost hysterical, and on Friday was much fatigued. During Friday night she had difficulty in swallow-

ing and by Saturday morning she strangled when she attempted to swallow. She coughed a great deal, but was unable to raise the thick, tenacious mucus from the pharynx. Articulation became difficult and speech was indistinct. There was persistent constipation after the initial diarrhea, and, except for the initial cramps, there was no pain. On account of the blindness no disturbance of vision was noted. The patient died Sunday night.

Necropsy was not obtained in either case. There was a history of both persons having eaten home-canned apricots which did not taste just right, but unfortunately the remnants of the apricots had been discarded. There was no record of the method used in canning the apricots or of the exact time at which the apricots were eaten.

OUTBREAK 11 (eight cases).—On Sunday, Jan. 27, 1918, a party consisting of nine persons, five adults and four children, had supper together near Madera, Calif. The supper consisted of fresh pork, brown beans, bread, butter, milk and home-canned apricots. It was noted that the apricots had a peculiar taste, but eight of the party ate some of them. The only member of the party who escaped illness was the one who did not eat any of the apricots.

On Tuesday morning, January 29, three of the children, aged 3½, 5 and 14 years, respectively, complained of seeing double, and three of the adults complained of dizziness. The three adults also developed diplopia during the day. The fourth adult first showed symptoms of illness Tuesday night, and the smallest child, aged 13 months, became ill Wednesday evening. One of the children died Wednesday morning, January 30, two Wednesday evening and one Friday morning. Two of the adults died during the night of Thursday and the remaining two have apparently recovered after a prolonged illness.

Drs. D. H. Ransom and St. J. Hely of Madera were called to care for the patients and it is to them that I am indebted for permission to study the cases, to perform necropsy in one case and to obtain various materials for bacteriologic examination.

The symptoms of all the patients were practically identical except in degree of severity. In all there were dizziness, weakness and incoordination of muscular movement, early disturbance of vision with blepharoptosis, mydriasis and diplopia, difficulty in swallowing and talking and strangling spells induced by attempts to raise thick mucus from the pharynx or to swallow. In one case, one of the patients who recovered, there was initial diarrhea and vomiting which occurred from twelve to fifteen hours before the onset of the eye symptoms, but in none of the other cases were there acute gastro-intestinal manifestations. In all the cases there was persistent constipation. The following case reports are very typical.

THREE CASE REPORTS IN DETAIL

CASE 1.—History.—F. M., aged about 60, had supper with the rest of the family on Sunday evening and ate some of the apricots. He worked all day Monday, although he "felt a little out of sorts," and slept well Monday night. On Tuesday morning he was very dizzy, and, later in the day, began to see double. There was free movement of the bowels after magnesium sulphate. He again slept well Tuesday night but on Wednesday morning was very weak and uncertain in his movements, although he remained up all day. He still complained of double vision and "blurring." On Thursday morning he was so weak that he was unable to dress. There was some vertigo. For the first time the patient complained of dryness of the mouth and pharynx, and during the day he began to have difficulty in swallowing and talking. Toward evening there was much difficulty in clearing thick mucus from the pharynx, and the patient began to have severe strangling spells when he attempted to swallow. He was very restless Thursday night and slept little, although there was no pain. On Friday morning he complained of being very hungry but said he was afraid to try to swallow. He complained

of being very weak, but was able to move himself in bed with little difficulty. There had been no bowel movement for three days.

Bedside Notes.—Physical examination was made by me on Friday about 10 a. m. The patient was lying comfortably in bed, apparently without pain. The temperature was 96.4 F., the pulse rate 72, and the rate of respiration 16. Respiration was not labored. Articulation was very much impaired; it was almost impossible to understand what was said, and the patient became quite irritable and stubborn when asked to repeat. He was able to raise himself in bed without difficulty and there was no apparent weakness of the muscles of the neck.

There was bilateral blepharoptosis, the pupils were dilated and reacted sluggishly to light, and the patient complained of photophobia and diplopia. There was no nystagmus. The mucous membrane of the mouth and pharynx was very dry and the tongue was swollen, heavily coated and fissured. The breath was very offensive.

Examination of the chest revealed nothing abnormal in the lungs. The area of cardiac dullness was slightly increased but the heart sounds were clear and the heart rate was not rapid. The blood pressure was 140 mm. systolic and 90 mm. diastolic (Tycos). The abdomen was relaxed and soft, there were no areas of tenderness, and no abnormal masses were felt. The skin was everywhere dry and somewhat atrophic.

Sensation was apparently unimpaired, mentality was clear, and the knee-jerks, plantar reflexes and biceps reflexes were normal.

Laboratory Examination.—The red blood corpuscles numbered 5,300,000 and the leukocytes 16,000, of which 88 per cent. were polymorphonuclear neutrophils, 9 per cent. were lymphocytes, 2 per cent. were mononuclears and 1 per cent. eosinophils.

The urine contained neither albumin nor casts, and microscopic examination of the sediment showed no casts, red blood cells, or other abnormal constituents.

Treatment.—On Friday afternoon feeding and administration of magnesium sulphate by stomach tube was commenced, and was continued until the patient was again able to swallow. Between Friday at midnight and Monday noon about 85 c.c. of immune goat serum were administered subcutaneously. (One c.c. of the serum was sufficient to neutralize approximately 3,000 minimum lethal doses of botulism toxin for a guinea-pig.) Supporting treatment was given throughout and a digitalis preparation and whisky were given when indicated. The necessary fluid was given by Murphy drip when the patient was unable to swallow.

Clinical Course.—For several days the patient appeared to be very ill, was unable to swallow, talk or control muscular movements, and was very weak. He was very restless and slept little. He was frequently irritable, but was always mentally clear. The temperature varied from 97.6 to 101 F. (by rectum) and the pulse rate was usually between 85 to 95 per minute.

By February 6 there began to be some signs of improvement. The patient was not so restless, he slept better, and at times was able to swallow small amounts of fluids. On February 10 he looked very much better and was able to talk more intelligibly. He stated that there had never been any pain except at the site of inoculation. The pupils were still dilated and still reacted sluggishly to light, but there was no diplopia. Bilateral ptosis persisted. The tongue was coated and very dry and fissured, but there was no longer an accumulation of mucus in the pharynx. Muscular coordination was improved, and the skeletal reflexes were normal.

Results.—On March 10, six weeks after the patient had eaten the apricots, he was able to be up, to walk without staggering and "to swallow fairly well." There was no ptosis, no dilatation of the pupils and no disturbance of vision. The voice was still husky, especially after a prolonged conversation, but articu-

lation was distinct. The patient became dizzy if he attempted to move quickly, but stated that he was rapidly growing stronger and more sure of himself. There was no evidence of any persisting paralysis.

CASE 2.—History.—C. M., aged 24, ate nothing but apricots, bread and butter for supper Sunday evening. He felt perfectly well on Monday, but on Tuesday afternoon he vomited and had diarrhea. On Wednesday he began to have double vision and dizziness, and he complained that when he tried to walk he staggered as if he were drunk. He was very restless and did not sleep well Wednesday night. On Thursday he was unable to leave his bed, and, although he was very hungry, he was unable to swallow any food. It was noted that enunciation was very difficult and indistinct.

Examination.—When seen by me on Friday, there was bilateral ptosis and some photophobia but the pupils were not dilated, and they reacted fairly actively to light stimulation. The skin was dry, the tongue heavily coated and the mucous membrane of the mouth and pharynx parched. There was no congestion of the pharynx. There were no signs of impairment in the lungs and the heart was apparently normal. There was nothing abnormal in the skeletal reflexes.

There was no demonstrable disturbance of peripheral sensation, and the patient was apparently quite clear mentally, although he became irritable when he could not make himself understood. Speech at this time was almost unintelligible.

The blood pressure was 135 mm. systolic and 90 mm. diastolic (Tycos), and the urine showed no albumin or casts. The red blood corpuscles numbered 5,600,000 and the leukocytes 19,000, of which 80 per cent. were polymorphonuclear neutrophils, 18 per cent. were lymphocytes and 2 per cent. were eosinophils.

Clinical Course.—At first it appeared as if the patient was much less severely poisoned than his father, but gradually he became much worse, due chiefly to the fact that he had a great deal of cough and many severe strangling spells. The temperature was as high as 101 F. on two occasions but usually was subnormal. The pulse rate was always above 80 and ranged between 90 and 115 for several days. On one occasion it reached 140 per minute.

Treatment.—This was the same as in Case 1. The same amount of anti-toxin was administered, food and laxatives were given by stomach tube and fluid was given by the Murphy drip method. A digitalis preparation was given as indicated.

Results.—Recovery in this case was very slow. On March 10, six weeks after the poisoning, the patient was still confined to bed and still was forced to take his food through a stomach tube. He had become greatly emaciated and was very weak, but muscular coordination was improved and there was no ptosis or diplopia. The skeletal reflexes were normal. There had been infection at the site of inoculation of serum and small pockets of pus were still present, although bacteriologic examination failed to show any pyogenic bacteria.

CASE 3.—History.—B. M., aged 13 months, ate very little of the fruit and did not show any symptoms of poisoning until Wednesday evening. At that time it was noted that there was bilateral blepharoptosis, mydriasis with little reaction to light, and apparent photophobia. There had not been any acute gastro-intestinal disturbance and the child did not appear to be in pain. The temperature was normal and the pulse rate 120.

Examination.—When seen by me on Friday, about two hours before he died, the child was apparently conscious but very apathetic. There was marked ptosis, the pupils were widely dilated and did not react to light and the skin was very dry. The heart sounds were normal and no dulness or râles were detected in the lungs. The skeletal reflexes were normal but the muscles were

all relaxed. The temperature was 99.2 F. (rectal) and the pulse rate 160. Death occurred from respiratory failure.

Necropsy Report.—Necropsy was done by me about four hours after death, and before the body had been embalmed. There was marked engorgement of the vessels of the pia mater and there were a few small hemorrhages on the surface of the pons, in the space between the cerebrum and cerebellum. There was no increase in the amount of fluid in the ventricles and the cerebral convolutions were not flattened. There was no general engorgement of the abdominal and thoracic viscera but there was beginning bronchopneumonia in the bases of both lungs. The spleen was not enlarged or soft.

Microscopic examination of the tissues showed no degeneration of the ganglion cells of the brain and cord and no thrombi in the blood vessels of the central nervous system. There were well marked patches of bronchopneumonia and many masses of cellular thrombus in the larger blood vessels of the lungs. Many of the smaller vessels contained hyaline masses in which were clumps of leukocytes.

Poisoning of the Domestic Fowls.—On Wednesday, April 29, the portion of the apricots which remained in the jar was thrown to the chickens. On Thursday several chickens showed symptoms of limber-neck and some of them died, and by Friday afternoon over twenty-five chickens and one turkey had died. A wild canary with similar symptoms was found lying under a tree and it died a few hours later.

Identification of B. Botulinus.—Bacteriologic examination of the contents of the gizzard of one of the chickens revealed the presence of a strain of *B. botulinus* which produces a virulent toxin when grown in suitable culture mediums. This strain, which we have labeled Strain 9, was shown to produce a toxin which is homologous with Toxins 3, 4, and 7 of my series.

Methods Employed in Canning.—We were unable to learn the method by which the apricots had been canned, but it is known that the fruit was unsalable, wind-fall fruit, and that Mrs. M. had marked the jars for early use as she feared the fruit would spoil. It was noted by some of those who ate the apricots that there was a peculiar sharp taste, but no unusual odor was detected.

OUTBREAK 12 (one case).—On Friday, Feb. 22, 1918, Mrs. H. of Oakdale, Calif., tasted a small portion of a bean pod from a jar of home-canned string beans which she had just opened. The beans had a peculiar irritating taste but she swallowed a small amount. When she placed the beans on the stove to cook there was an extremely disagreeable odor and she discarded them.

On Saturday morning, February 23, Mrs. H. was dizzy when she first got out of bed and she staggered when she walked. She noted that vision in the left eye was blurred. By afternoon vision was blurred in both eyes and the staggering was much worse. The patient became alarmed and consulted Dr. J. B. Thompson of Oakdale, to whom I am indebted for the following report.

When first seen by Dr. Thompson, Mrs. H. was almost hysterical but had no complaint other than that which has been detailed. She said that there had been no pain and no gastro-intestinal disturbance, and that she had not even been nauseated.

Examination showed complete dilatation of both pupils, which reacted very sluggishly to light, and a pulse rate of about 100 per minute. The temperature was normal. The patient was very weak and muscular movements were not coordinated, but nothing else abnormal was found. Dr. Thompson stated that he was strongly suspicious of belladonna poisoning, as at that time the patient denied having eaten any spoiled food.

After midnight, Saturday, Mrs. H. complained that her tongue was swollen, and she began to have difficulty in talking. On Sunday morning there was bilateral ptosis and later in the forenoon she began to have difficulty in swallowing. Speech rapidly became unintelligible. About noon on Sunday she

began to vomit and the vomiting continued for the greater part of the afternoon. There were three formed stools during Sunday forenoon and several involuntary liquid stools during the afternoon. All the stools were extremely offensive. There were no bowel movements during the last thirty-six hours of life. Toward evening on Sunday the patient began to have spells of strangling when she attempted to swallow, and she complained bitterly of the accumulation of mucus in the pharynx, which she was unable to raise. Urination was not disturbed except that the patient voided involuntarily during strangling spells.

The temperature varied from normal to 102.6 (rectal) just before death, respiration remained about 24 and the pulse varied from 98 to 120 per minute.

The patient was very restless and did not sleep during Sunday night or Monday. There was no disturbance of mentality and the skeletal reflexes were intact. There was no disturbance of sensation except a sense of numbness in the hands. She became rapidly weaker and the pulse increased in rapidity. At first there was satisfactory response to stimulation but later there was no appreciable effect. The patient died early Tuesday morning, about eighty-eight hours after tasting the beans.

To remove the mucus from the pharynx, Dr. Thompson made use of an aspiration bottle to which a soft rubber catheter was attached. He stated that this appeared to give much greater relief and to cause much less irritation than swabbing.

The beans which caused the poisoning were grown in Oakdale and were picked but a few hours before they were canned. They were washed, strung, broken into small pieces and boiled in an open kettle for twenty minutes. They were then packed into new, freshly-boiled, 1-pint economy jars, 1 tablespoonful of salt was added to each jar and the jars were covered, immersed into boiling water, which covered them, and boiled actively for three hours. Twelve jars of beans were canned and two of them spoiled. The contents of nine of the others were eaten after they had been cooked, and appeared to be good, but there is no record as to whether any one had eaten or tasted any of the beans before they were cooked. One jar was taken to the laboratory for bacteriologic examination, but no evidence of *B. botulinus* was found.

A portion of the beans from the contaminated jar was gathered from the garden where they had been thrown and had lain in the rain for several days. Bacteriologic examination showed no evidence of *B. botulinus*.

OUTBREAK 13 (one case).—On Sunday, Feb. 17, 1918, Mrs. K. of Los Angeles, opened a jar of string beans which she had canned during the past summer. She tasted four of the beans and noted that they had a peculiar taste which she at first attributed to the presence of some lemon juice which had been added to the beans when they were canned. She eventually decided that they were not good, and discarded them.

On Monday morning Mrs. K. "was distressed" and took a small dose of Epsom salts. Toward evening she vomited, and then took a large dose of Epsom salts, after which the bowels moved several times. On Tuesday she was worse, and consulted Dr. Thomas C. Myers of Los Angeles who sent me the record of the case.

When Dr. Myers first saw Mrs. K. she was extremely nervous and complained of double vision and of "swelling in the throat," dryness of the mouth and difficulty in talking and swallowing. There was marked muscular weakness and bilateral ptosis but the pupils were not dilated. The temperature was subnormal and the pulse was rapid. The patient died early Wednesday morning, about sixty hours after she had tasted the beans.

A portion of the discarded beans was recovered from the garbage can, several days after they had been discarded, but bacteriologic examination failed to show the presence of *B. botulinus*.

The beans were canned by Mrs. K. during the past summer. The exact method of canning could not be obtained, but it was known that the beans were washed, broken and packed into 1-quart, self-sealing Mason jars, and

that 1 teaspoonful of salt and "a small amount" of lemon juice were added to each jar. The jars were filled with cold water and the covers placed in position, after which they were placed in a wash-boiler and "steamed for three hours." It was impossible to find out whether the jars had been immersed in boiling water or whether they were merely steamed, nor could we learn whether the three hours was estimated from the time the jars were placed in the wash-boiler or from the time the water commenced to boil.

OUTBREAK 14 (one case).—On Sunday, March 10, 1918, Mr. A. Y. of Colton, Calif., ate home-canned pears which had a peculiar taste. On Monday afternoon he complained of a sensation of swelling of the tongue, and of vomiting. In the evening he consulted Dr. J. S. Champion of Colton, who sent me the record of the case.

Mr. Y. complained of indistinctness of vision, diplopia and vertigo and stated that he was unable to walk straight. There was some difficulty in swallowing and in talking, articulation being so indistinct that it was difficult to understand what was said. There was ptosis of the left eyelid.

There was moderately severe constipation although the bowels moved freely after the administration of Epsom salt. Diplopia and vertigo persisted for over three weeks, but after a few days the patient was able to swallow liquids, although it was impossible to swallow solids. At first there was some annoyance from an accumulation of thick mucus in the pharynx but this did not persist. The temperature ranged between 97.6 and 99.6 F., and the pulse between 60 and 112. It is interesting that the pulse rate gradually increased from 60, the rate when the patient was first seen, to 112 on the third day.

Mentality was clear throughout, but this case is of interest in that there was a persistent numbness of the left side of the body. At the end of three weeks the patient was much improved, although there were still some eye symptoms and marked weakness.

It was not possible to determine beyond a possibility of doubt that the pears were the cause of the poisoning since it is not known whether other persons partook of the same pears without suffering ill effects. Neither was it possible to learn the method which was adopted in canning the pears, but there was no record of the patient having eaten any other food which gave any indication of being spoiled.

B. REPORT OF INSTANCES IN WHICH DOMESTIC FOWL AND ANIMALS WERE POISONED BY FEEDING SPOILED, HOME-CANNED VEGETABLES

In our investigation of outbreaks of food poisoning caused by home-canned products we have obtained records of at least ten outbreaks of poisoning of domestic animals and fowl caused by feeding spoiled, home-canned vegetables. These are in addition to ten other instances in which the poisoning of the animals and fowl occurred coincidentally with outbreaks of poisoning of human beings, the animals and fowl having been poisoned by feeding them remnants of the food which had caused the poisoning of the human victims, and to several other instances in which it was believed that the poisoning was due to feeding spoiled home-canned products, although we were unable to obtain sufficiently accurate details to justify making a definite conclusion that such was the case. Six of these ten outbreaks were caused by feeding spoiled home-canned string beans, three by feeding spoiled home-canned corn and one by feeding spoiled home-canned green peas.

CAUSE OF THE POISONING CONFIRMED

The reasons for assuming that these outbreaks of poisoning of domestic animals and fowl were dependent on the presence of the toxin of *B. botulinus* in the spoiled home-canned products are the following: (1) The symptoms were identical with those which occurred in animals and fowl which had been poisoned by food which had caused typical botulinus intoxication of human beings, and in which *B. botulinus* was recovered from remnants of the discarded food; (2) it has been shown that symptoms identical with those which were observed in these animals and fowl may be produced experimentally by feeding the toxin of *B. botulinus* to domestic animals and fowl,³ and (3) in one instance recorded in the following (Outbreak 1) we were able to demonstrate the presence of botulinus toxin in remnants of the mash which was fed to the chickens and to recover *B. botulinus* from the crops and gizzards of two of the chickens which died as well as from three out of six jars of the same lot of string beans which remained in the vegetable closet.

Four instances of poisoning of domestic animals and fowl are detailed because of the fact that the methods by which the vegetables were canned have been ascertained.

CASES OF POISONING IN DOMESTIC ANIMALS

OUTBREAK 1.—On Feb. 12, 1918, Mrs. M. of Berkeley, Calif., opened a jar of home-canned string beans and noted that they had an unpleasant odor, "something like cheese." She mixed the beans with a mash which she was preparing and fed them to the chickens. Eleven of her twelve chickens developed typical symptoms of limber-neck and died.

Six jars of beans which remained in the vegetable closet and the carcasses of several of the chickens as well as a portion of the mash which remained over, were taken to the laboratory for examination. From the crops and gizzards of two of the chickens and from three of the jars of beans a virulent strain of *B. botulinus* was recovered, and in the mash it was found that there was still a virulent toxin present. The toxicity of the beans in the jars was comparatively high, 0.001 c.c. of the filtered bean juice being sufficient to kill a 250 gm. guinea-pig within twenty-four hours. This toxin was shown to be homologous with that of Strain 6 of my series, thereby differing from all other strains which have been isolated on the Pacific Coast.

The beans were grown by Mrs. M. in her own garden and were picked but a few hours before they were canned. They were washed, broken into small pieces and packed into 1-quart Mason jars. The beans were covered with cold water and were sterilized by leaving the jars immersed in boiling water in a wash-boiler for two and one-half hours on each of three consecutive days. Mrs. M. does not remember whether the jars were sealed during the intervals

3. Dickson, E. C.: Botulism: A Cause of Limber-Neck in Chickens, Jour. Am. Vet. Med. Assn., 1917, **50** (New Series III), 612. Buckley and Shippen: Preliminary Report on the Relation of Anaerobic Organisms to Forage Poison—Am. Vet. Med. Assn., 1917, **50** (New Series III), 612. Buckley and Shippen: Preliminary Report on the Relation of Anaerobic Organisms to Forage Poisoning, *ibid.*, 1917, **50** (New Series III), 809. Graham, Brueckner and Pontius: Studies in Forage Poisoning V and VI, Kentucky Agricult. Exper. Sta., Bull. 207, 1917.

between sterilizations, as she had been told that it made no difference whether they were sealed or left open.

OUTBREAK 2.—On Feb. 15, 1918, Mrs. L., near Hollister, Calif., noticed that one of her hogs was dragging its hind legs and that it could not eat. On the following day the hog was down and could not get up. When disturbed it tried to squeal but was unable to do so. It remained in the same position for several days and it was noted that there had been no evacuation of the bowels during that time.

About three days later another hog became affected in the same way and Mrs. L. called in Dr. C. S. Brooks of Hollister, the county veterinarian. It was through the courtesy of Dr. Brooks that I was permitted to see these animals.

The symptoms of both hogs were the same; they first dragged their hind legs, eventually went down and could not rise, were unable to eat although they tried to do so, were unable to squeal and were constipated. The pupils were dilated and did not react to light, the temperature was normal and heart beat rapid. Both animals were given Epsom salt with good results. Both remained in the same condition for about one week and then gradually recovered. Within two weeks they were able to move around, to squeal and to eat.

It was learned that the hogs had been fed the contents of a jar of home-canned green peas which had spoiled and which had a very offensive odor. None of the peas were fed to chickens or to other animals.

The peas had been grown by Mrs. L. and had been placed in 1-quart Mason jars and sterilized by immersing the jars into boiling water in a wash-boiler for three hours. All Mrs. L.'s vegetables had been canned by the same method and there had been a considerable amount of spoilage.

There may be some doubt as to whether the illness of the hogs was really due to poisoning with botulinus toxin as hogs are supposed to be immune to botulinus intoxication. However, the symptoms were identical with those which we have produced experimentally by subcutaneous injections of the toxins of our Strains 3 and 4 of *B. botulinus*, and a virulent strain of *B. botulinus* was recovered from a jar of spoiled string beans which was canned in the same way and which remained in Mrs. L.'s cellar.

OUTBREAK 3.—During the summer of 1917, Mr. H. of San Jacinto, Calif., canned several jars of corn by the fractional method of sterilization, in which he closely followed the instructions given in U. S. Department of Agriculture Farmers' Bulletin No. 359. About one month later he noticed that some of the corn was spoiling and that there was gas formation. He discarded the whole lot and fed it to the chickens. Within from twelve to twenty-four hours a number of his chickens developed typical symptoms of limber-neck, and within two or three days fifty-eight out of sixty-six died. Mr. H. stated that he had no way of knowing whether the eight survivors had eaten any of the corn.

It was not possible to obtain material for bacteriologic examination.

OUTBREAK 4.—In January, 1918, Mrs. L. of Raymond, Calif., threw out five jars of home-canned food which had spoiled, three 1-quart jars of peas, one 1-quart jar of string beans and one 1-pint jar of beef. The contents of all the jars had an offensive odor and were not eaten or tasted by human beings.

Within a few hours after eating the discarded food, eighty-one chickens developed typical symptoms of limber-neck and seventy-two of them died in from one to three days. Mrs. L. stated that the survivors had each been given a tablespoonful of castor oil.

The food was canned by Mrs. L. who stated that very little of her canned goods had spoiled. The material was packed in 1-quart Mason jars which had been boiled, and was cooked and sterilized by immersing the jars into boiling water in a wash-boiler for three hours.*

* This outbreak is not included in the series of ten recorded as being due to home-canned vegetables, as there is a possibility that the meat was at fault.

COMMENT

A study of these cases as well as of those described in my previous report¹ fully justifies the conclusion that the use of home-canned food is not wholly unattended with danger. In the instances which have been cited, various methods of canning were adopted and were followed as carefully as it is possible to have untrained persons conduct technical procedures. The fact that in several instances the greater part of the home-canned material remained in good condition simply proves that though the methods are efficient in preventing ordinary spoilage, they are inefficient if the raw material happens to be contaminated with spores of *B. botulinus*. It has been stated that it is essential that only freshly picked raw material should be used for canning and that it should be blanched before it is placed in the jars, but Outbreak 12 shows clearly that three hours' sterilization in the washboiler is not sufficient to kill spores of *B. botulinus* in vegetables which had been freshly picked and had been boiled for twenty minutes in an open kettle before being packed into 1-pint jars, a procedure which should be at least as efficient as simple blanching.

These cases also illustrate the fact that it is unsafe to eat or even taste home-canned products before they have been boiled. In six outbreaks of botulism the victims were poisoned by simply tasting home-canned goods to determine whether they had spoiled, and in several others the poisoning was caused by the ingestion of salads prepared from uncooked, home-canned vegetables. It is well known that the toxin of *B. botulinus* is quickly destroyed by heat and Outbreak 8 is an illustration of this fact, since those who ate the cooked asparagus on Sunday night remained in perfect health, whereas the man who ate the remainder of the asparagus from the jar, uncooked, on Monday night, developed the typical symptoms of botulinus intoxication.

It is not the object of this report to discourage the home-canning of fruits and vegetables or to interfere in any way with the active campaign for the conservation of perishable foods. It is of the utmost importance, however, that those who are directing the home-canning industry should recognize that the present methods of home-canning are not entirely safe, especially in the hands of untrained workers. If they will but admit this fact and will instruct the public that there is possible danger of poisoning from home-canned products, and that the danger may be averted if all home-canned food is boiled before it is eaten or even tasted, outbreaks of botulism from home-canned products will entirely cease.

Sacramento and Webster Streets.