BOTULISM

Its

DISTRIBUTION, CAUSE and PREVENTION.

By

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BOTULISM.

Botulism is a specific food-born intoxication caused by the toxin of the Bacillus botulinus. The term is derived from the word 'botulus,' meaning a sausage; for the disease was most frequently associated with eating of sausage in Germany. The organism is essentially saprofitic, growing in a great variety of foodstuffs, both of animal and plant origin. It produces its poison in the food before it is eaten.

The disease occurs more commonly in Europe than in America. Thelargest number of cases are reported from Germany and Russia, probably because of the large amount of raw meats consumed in those countries in the form of sausage. No cases are reported in England, Wales or Scotland. It has a high mortality and distressing symptoms; and its relation to food makes it dramatic news, and these facts have recently caused concern and alarm out of proportion to the prevalance of the disease. One hundred fifty cases of the disease with one hundred eleven deaths among one hundred millions of people in twenty-two years cannot be compared in magnitude to tuberculosis, syphilis and other public health problems.

It has no relation to sex, age, season, or social condition. It is also one of the causes of forage poisoning in horses, limber-neck in chickens and turkeys, and possibly causes paralysis in some domestic animals.

The symptoms appear in from 18 to 36 hours after ingestion of the poisonous food. Cases have been reported with incubation periods of four hours, and others of six days. The incubation period depends on the amount and virulence of the toxin. There are no gastro-intestinal symptoms as a rule, but these may appear. ~~The~~ The first symptoms are indisposition, weakness, fatigue, early

disturbances of vision, blepharoptosis, dilatation of pupils, and loss of reflex to light. Loss of accommodation is soon complete. Constipation is almost constant, and soon there is difficulty in swallowing and talking, ~~and~~ the throat becomes dry and there is thirst. There is progressive muscular weakness, but almost complete absence of sensory disturbances. There is usually no pain and the mind is clear. The pulse is rapid, and the temperature subnormal. Fever coming on late indicates bronchopheumonia. Death is due to paralysis of respiration. The mortality is high, sometimes 100%, and in other outbreaks only 10%. The average is about 45% to 50%. There is marked congestion and some hemorrhage of the central nervous system, and the lungs are hyperemic. Food has been known to remain in the stomach for several days in cases of Botulism, and for this reason it is important to wash out the stomach and intestine as soon as the diagnosis is made.

The Bacillus botulinus was discovered by Van Ermingen in 1895, in some pickled meat, in Germany. It is a large, slightly motile, Gram positive rod, with four to eight flagellae, and has a large spore at one end. It grows at room temperature best, but will grow at body temperature, and is anaerobic in nature. It is strongly proteolytic. Gas is formed due to fermentation of sugars. There are non toxic strains of the organism as well as those which are toxic. The nontoxic forms must be differentiated from Bacillus sporogenes.

Strains A and B of the organism are recognized which produce specific toxins and antitoxins. It is believed by many that the bacillus is associated with animal manure and that the spores are widely distributed in soil and dust. Recent investigations by Easton and Meyer(1) have shown that the organisms and spores are widely

distributed, and that in some cases the type A is found in animal excreta (5 out of 50 samples examined), but also that animal excreta has little to do with the spread of Bacillus botulinus. These investigators failed to find the organisms in the stools of 88 healthy persons, altho it was known that the spores were being ingested on raw fruits and vegetables which served as food. The presence of botulinus toxin, type B, however, was demonstrated in cultures inoculated with a specimen of feces from a typical case of botulism taken on the sixth day of the disease.[2] A pure culture of B. botulinus, type B, was readily isolated from the cultures in which toxin had been demonstrated, by the use of 5% horse blood extract agar plates, incubated in a hydrogen-anaërobic jar. This method is of value as bacteriological evidence when a specimen of suspected food cannot be obtained.

The organism has been found in many samples of soil from various parts of the United States, particularly in the mountainous regions. Along the Atlantic Coast the type B seems to predominate, while in the West Type A is most often found. Stricker, Levin and Benson[3] state that the spores of Clostridium botulinum have been found in 31% of the soils examined from Oregon, 80% from Washington, and 29% from California. The outbreak in Albany, Oregon in 1924 was one of the largest that has ever occurred in this country. Every person (12) who ate at this meal suffered death from the virulent toxin that was present in a jar of string beans. The history of the outbreak, from a preventative standpoint, emphasizes the fact that a great deal of care must be exercised in canning methods. The soil of the garden in which these beans were grown was found to contain Clostridium botulinum, type A.

Weiss[4] has determined in the laboratory of M. J. Rosenau[4] that for the botulinus spores formed under the most favorable conditions for survival, the most resistant types require five hours at boiling temperature, 45 minutes at 105°C., 15 minutes at 110°C., and six minutes at 120°C. to cause their destruction. Young spores are more resistant than old, and dry ones are more resistant than moist, and acids, ~~and~~ alkalies and various chemicals greatly diminish the thermal resistance.

Dozier[5] asserts that 10% hydrochloric acid at room temperature may be depended on to destroy all B. botulinus spores within an hour. If a longer time can be allowed for sterilization, liquor formaldehydi diluted once with warm water acting over a period of at least 24 hours is recommended. It is quite probable that such a solution held at from 60° to 70°C. for a shorter period would be efficient.

The organism produces a true, soluble, exotoxin comparable in all respects to the poisons produced in cultures of Diphtheria. It is the only one of the true toxins which ~~are~~ is poisonous when taken by mouth. It is pathogenic to guineapigs, mice, and monkeys as well as to man when taken in this manner. It is very potent,--one or two drops of a culture taken on a piece of bread causing death in a few days. As little as 0.000,001 cc. of some toxins will kill a 250 gm. guineapig in three or four days.

The toxin is produced under anaërobic conditions by the bacillus grown on suitable media. There are some strains which do not produce toxins, and the property may be lost by others when long cultivated on artificial media. The evidence presented by Dozier[6] seems to suggest that toxin production by B. botulinus is a function of enzyme action, and the bacillary cell is probably the

matrix of the poision. The optimum temperature for toxin production is 37°C. but it is produced also between 20° and 30°C. It is not formed in brine containing over 6% of sodium chloride. This is of importance in pickling foods.

It has been shown by Van Ermengem that the toxin is destroyed by heating at 80°C. for one-half hour. Others have confirmed this and have shown that it carries a wide margin of safety. According to Orr, the most resistant of ten strains studied by him was destroyed at 80°C. for two minutes, 72°C. for ten minutes, and at 65°C. for 85 minutes. The toxins of most strains are destroyed in 30 minutes at 65°C. It is, therefore, destroyed by cooking. The outstanding findings of clinical improtance made by Schoenholz and Meyer[7] is that suspicious food, altho thoroly cooked, is not fit for human or animal consumption. The toxin in solution is very resistant to light and air, and is not affected by drying or putrefaction. It has also been observed in the course of experimental work that ethyl alcohol destroys the toxin of B. botulinus.

Rosenau states in the Nelson Loose-Leaf Medicine, that the toxin has a special affinity for the central nervous system:--that it is almost a pure neurotoxin; and that its effect is mainly on the central nervous system, but it also acts upon the blood vessels, causing dilatation, thrombi, and hemorrhage. Seale Harris in the Tice Practice of Medicine[14], declares that the symptoms are due to degenerative changes in the central nervous system; particularly the motor cells of the medulla and spinal cord. In contradiction to this, Cowdry and Nicholson[8] believe, as a result of more recent work, that except fot a slight degree of vascular engorgement, no brain lesions are caused by the direct action of botulinus toxin. Their observations tend to show that it produces a curara-like paralysis and that the site of action of the toxin is on the

peripheral nerve terminals. This belief is also held by Sisco[9] in a report given in November 1924.

A true antitoxin may be produced in animals by injecting increasing amounts of toxin into susceptible animals. This antitoxin has both preventative and curative values if given before the onset of the symptoms. The antitoxins for strains A and strains B are specific for those strains of the toxins only; therefore both strains of antitoxins must be available in order to be of practical use. If chickens which have eaten refuse from the kitchen develop limberneck, it is an indication to administer antitoxin to those who have eaten at the meal from which the refuse came. Chickens usually develop the symptoms within a few hours and die within 24 hours, and in many instances this will be in plenty of time to administer the antitoxin to the human beings, as they may not develop the symptoms until after 24 hours. In order to be effective antitoxin must be given before symptoms appear.

Experiments performed by Wemberg and Goy[10] showed that immunization of animals is easily induced thru injection of botulism antitoxin long acted on by formaldehyd solution. The immunity is pronounced; the blood serum flocculates in the presence of a corresponding toxin, and the antitoxic power is high. But the action of the formaldehyd decreases the duration of the immunity.

The presence of animal protein is not necessary for the growth of botulinus, as was formerly thought. In Europe the foods usually containing the toxin have been sausage and hams, but in America string beans, cottage cheese, corn, asparagus, spinach and ripe olives have all been offenders. Chicken, turkey, beef and fish have also been culture media, and caused poisoning. It is

often difficult to determine the food causing the disease as the symptoms rarely appear before 18 to 36 hours, and several meals have been taken since the poison food was eaten. If nausea and vomiting are the first symptoms, the food vomited is often accused of causing the distress, when it really is not.

Home packed foods have been especially responsible for outbreaks of the disease, because the temperature used is often not sufficient to kill the botulinus spores which may contaminate the food at the time it is packed. A large number of the ripe olive cases have been due to the fruit put up in glass and improperly processed. An outbreak in Darmstadt, in 1904, was caused by a salad prepared from home canned white beans. Sausage is the most frequent source of botulism in Germany. Odor, appearance, or taste of the food cannot be relied upon, as it may contain the toxin and still to all outward indications be perfectly safe. But the toxin is most frequently associated with some decomposition as well. Merely tasting or nibbling the contaminated food has proven fatal.

The three outbreaks of botulism due to canned ripe olives early in 1924 revived suspicions aroused by the outbreaks of 1919 and 1920. A survey made by the Bureau of Chemistry, of the Department of Agriculture,(11) in which over 800 samples(in 2900 containers)of spoiled or suspected materials,found on shelves of dealers in 30 different states, District of Columbia and Porto Rico, were examined, showed that canned ripe olives which were free from swell, from abnormal odor, or other marks of spoilage were not dangerous. B. botulinus was not found in any samples of this type. The investigation furnished no ground for attributing special danger to the consumption of canned ripe olives, if resonable care is exercised to exclude spoiled products from sale or use. Great care must be

exercised in the avoidance of unfit material.

There have been 134 outbreaks of botulism in the United States[9], involving 458 people, of whom 309 or 67% died. Many cases recover spontaneously without any treatment and cases showing definite symptoms havebeen reported as recovering. There is direct relationship between the period of incubation and the amount and virulence of the toxin ingested.

Outbreaks of the disease are appearing more frequently in recent years, probably because of the increased use of canned foods by city people, particularly the home canned foods. The pressure cooked factory canned foods are safe, and undamaged cans of this type are probably never causes of the disease.

Dickson reports an outbreak in which he isolated the bacillus from home canned beans which had caused 12 cases in a dormatory at Stanford University. Cooper[12] reports three cases occurring in one family as a result of eating beans salad made from home packed green beans mixed in some sliced onions, and mayonnaise dressing. The can had not been heated before it was opened.

From these reports and facts it can be concluded that the disease of botulism is produced by a toxin which is produced in food before it is eaten and is ingested with the food; that it is wide spread over the world; and that it can be prevented by using proper care in the preparation of foods, being sure that suspected foods are thoroughly cooked, better under pressure; and that the disease may be prevented if the corresponding antitoxin is given intravenously before the symptoms appear in case someone is unfortunate enough to eat some contaminated food. This last measure necessitates the identification of the organism and the determination

of its type.

One of the greatest obstacles to the treatment of botulism is the limited distribution of antitoxin.(9) The scarcity and sporadic nature of the outbreaks render the commercial preparation of antitoxin unprofitable, and it can now be obtained only at institutions where studies of botulism are being made.

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