MEAT POISONING AND THE MEAT-POISONING BACILLI.*

By GEORGE F. BUCHAN, M.B., Ch.B. (Glas.),
D.P.H. (Camb.).

Assistant Medical Officer of Health, City of Birmingham.

ALTHOUGH doubtless many outbreaks of meat poisoning have occurred in the centuries that are past, it is only forty-one years ago now since the first outbreak was put on record. This outbreak occurred at Fluntern in Switzerland, and twenty-seven persons are reported as having been seized with the symptoms of meat poisoning after eating veal. The calf from which this veal had been derived was very young—only five days old—and its joints contained what is described as “yellow water.” Probably it was suffering from some form of septicæmia. Since that time our knowledge has made rapid progress, and probably Bollinger was the man who gave the earliest attention to the subject. It was he, at least, who, in an address delivered in 1876 before the German Society for Public Hygiene at Düsseldorf, first called the attention of the medical world to the great importance of septicæmia and pyæmia in our food animals as a cause of meat poisoning. He states that, from the point of view of human health, these diseases are relatively of greater consequence than anthrax and glanders, since, in the first place, they occur more frequently, and in the second, their toxins are more resistant. This latter statement is remarkable in the light of later evidence, as Fischer has stated that the toxins of the *Bacillus enteritidis* of Gärtner, now proved to have been the cause of numerous outbreaks, will resist a temperature of 100° C. for one and a half hours; and Cathcart has supported this to some extent by experiments carried out less than two years ago, in which he demonstrated the virulence of these toxins after exposure to 100° C. for thirty minutes. In 1880, Bollinger again refers to the subject of meat poisoning, reasserts his views stated four years earlier, and affirms that careful observations in eleven extensive outbreaks of meat poisoning, with 1,600 cases, have amply justified his earlier conclusions. In this latter address he makes a critical survey of the literature on the subject up to 1880. This is extended by

Ostertag, in his handbook on meat inspection, up to the year 1903. During these thirteen years there are to be found records of ninety outbreaks, in which 4,000 cases occurred, which are attributed to the ingestion of meat from unhealthy animals. Septicæmia or enteritis in one form or another is named as the disease from which these animals suffered.

Of all these outbreaks the most important from the etiological point of view is that which occurred in Frankenhausen in May, 1888, and which was investigated bacteriologically by Gärtner. From a fatal case and from the incriminated meat he succeeded in isolating the bacillus which now goes by his name as the *Bacillus enteritidis* of Gärtner. Although the cow from which the meat was derived had suffered from persistent diarrhoea some time before death, the meat appeared perfectly good, and the spleen, liver, and other organs appeared normal, and all that could be detected was a slight reddening of the intestinal mucosa. Next perhaps in order of etiological importance was the outbreak in which the etiology of sausage poisoning, botulism or allantiasis engaged the attention of Van Ermengem. Until this time the symptoms of sausage poisoning were held by the scientific world to have been caused by the various products of the decomposition or putrefaction of proteids. But on the occasion of an outbreak of this disease at Elezelles in December, 1895, Van Ermengem, after prolonged investigation, isolated the *Bacillus botulinus* from portions of a ham which had caused the deaths of three persons, and symptoms of sausage poisoning, more or less severe, in seven others; and to this bacillus he attributed all the symptoms.

Since those dates bacilli belonging to those two classes have continued to hold the field as the etiological factors in cases of food poisoning. The *Bacillus botulinus* is in a class by itself, but many members—about thirty—of the enteritidis group have been described.

The pioneers in bacteriology, and especially those who worked on the nature of the substances secreted by bacteria, very early discovered the existence of what they called the ptomaines. These bodies have many of the chemical and physical properties of the vegetable alkaloids. Long before this discovery, observers had noted that the symptoms manifested by sufferers in many outbreaks of food poisoning were similar to those produced by certain vegetable alkaloids. It is not surprising,

* A Paper read before the Midland Branch of the Incorporated Society of Medical Officers of Health on March 19th, 1908.

therefore, that on this evidence the conclusion was arrived at that food poisoning was the result of the ingestion of these ptomaines, and that food poisoning and ptomaine poisoning were synonymous terms. Strictly speaking, the term ptomaine poisoning is not of such general application, and should be used only in those cases where the symptoms are the result of the ingestion of ptomaines preformed in the food. It is, however, a very grave question if ever a case of food poisoning occurred under those conditions. The quantity of ptomaines which can be demonstrated in decomposing food stuffs is always extremely small. The liquid containing the ptomaines, in food under examination, is always more deadly in its effects than the isolated ptomaines; and very many ptomaines have been isolated which are practically without effect on the human system. Brieger it was who first discovered the ptomaines more than twenty years ago now. Since then bacteriologists have not been idle, and they have demonstrated that there are bacterial products which are much more potent than the ptomaines, or, for that matter of it, than any known alkaloid. I refer to the toxins. These bodies have not all the same empirical formula, nor are they all of the same nature. They differ widely. Some belong to the class of ferments. Others resemble albumoses and proteoses—bodies of complex molecular constitution, formed at an early stage in the digestion of proteid. Toxins may be divided into two classes with reference to their seat of formation :—

1. "Intra-cellular toxins," contained within the bodies of the organism; and
2. "Extra-cellular toxins," produced in the culture medium.

Some bacilli may develop both kinds of toxins which may be recognised by their different properties. In considering, then, the etiology of food poisoning, we must bear in mind that these toxins may play a part. We may describe as "intoxications" those cases which result from the ingestion of food containing these toxins, and these toxins alone. But this condition would only hold where the bacteria had died out, their extra-cellular toxins, if any, remaining in the food, and their intra-cellular toxins, if any, remaining in the dead bodies of the bacteria. It will take many, many days, and often many, many weeks, for bacteria to die out in a medium in which they grow and multiply; and long

before this has occurred, the food, you will agree, would not be in such a condition as to tempt even the most hungry. On the other hand, in all recorded cases of food poisoning the food has not been in an obviously bad condition; and the point which I desire to make is that living bacilli gain admission to the body in cases of food poisoning just as much as in cases of typhoid fever and diphtheria.

The chief point in the diagnosis between poisoning by toxins and infection by bacteria is cited time and again as the length of the incubation period, a short incubation period pointing to the former, and a long to the latter. But surely it is not necessary to pre-suppose a chemical agent when we have a rapid onset of symptoms. If it is, the whole etiology of disease will require to be remodelled. In my view, it is the dose and virulence of the organism and the susceptibility of the patient which determine the length of the incubation period, as well as the severity of the attack. Of course, no one is in a position to deny absolutely that the ingestion of toxins, and toxins alone, may produce the symptoms of food poisoning, but up to the present time we have no information that such cases occur. On the other hand, we can clearly state that in all cases where a complete bacteriological examination has been made, bacteria, as the causal agents, have been isolated from the organs of fatal cases, thus proving that an actual infection by living organisms had occurred.

What, then, are the various microbes concerned with these outbreaks? I have already indicated that the vast majority belong to the enteritidis group, and the other is the *Bacillus botulinus*, an obligatory anærobe isolated from a sample of ham by Van Ermengem in 1895, by Romer also from ham, while Kempner isolated it from the intestine of the pig. By far the more important is the enteritidis group, and to it I propose to give the greater share of attention this afternoon. It contains a group of organisms of the very greatest interest and importance, which may be considered as intermediate in their characters between the coli group on the one hand and the typhoid group on the other. But, after all, the members of these groups are readily distinguishable. The bacilli of the enteritidis group are actively motile, non-sporing organisms which do not retain the stain by Gram's method. Their growths on agar and gelatine slopes are creamy and trans-

lucent, and the latter medium shows no liquefaction. Their fermentation tests are important, and to some extent diagnostic. They do not ferment lactose—and this at once distinguishes them from the coli group—or saccharose. They all ferment glucose, mannite, laevulose and dulcitol. But their most characteristic reaction (with one exception—*Bacillus paratyphoid A*) is that with litmus-milk, in which they produce transient acidity, followed by distinct and definite alkalinity. This is a very curious reaction, and chemically it is difficult to understand. It can only be attributed to the ultimate formation of amines, which must be formed from the nitrogenous compounds of milk in relatively greater quantities than the lactic acid from the lactose, which accounts for the first part of this phenomenon. The time taken for this reaction varies with the organism. The exception, *Bacillus paratyphoid A*, produces permanent acidity. Indol, when it is produced at all, is very slight, hardly recognisable.

The group may be divided into three sub-groups:—

1. The Gärtner sub-group, including the *Bacillus enteritidis* of Gärtner.
2. The Aettrycke sub-group, including the bacillus of hog cholera, which Theobald Smith does not recognise as essentially different from the *Bacillus enteritidis* of Gärtner.
3. The Paratyphoid sub-group, including the *Bacillus paratyphosus A* and *Bacillus paratyphosus B*. These are the organisms which are of etiological importance in cases of paratyphoid fever. Most of the recorded cases have been attributed to the *Bacillus paratyphoid B*.

In addition to these there is the *B. psittacosis*, which was found in a disease of parrots, and was held to be responsible for an outbreak which occurred in France, and in which the symptoms were similar to those of food poisoning.

In order to distinguish these sub-groups it is necessary to resort to agglutination tests, their cultural reactions being practically alike; and for the satisfactory and complete identification of any of these organisms it is necessary to test them against sera prepared with known cultures, the limits of agglutination of these sera being known. For example, in the

particular outbreak of food poisoning due to eating brawn in which I was concerned, and which I reported fully in the *Lancet* of December 7th last, I isolated a bacillus with all the cultured characteristics of the *Bacillus enteritidis* of Gärtner. Still, it might have belonged to the hog cholera sub-group or the paratyphoid sub-group, whose cultural reactions are practically identical with those of the *Bacillus enteritidis* of Gärtner, and that it belonged to the Gärtner sub-group could not be averred until I had tested its agglutination with sera prepared with known cultures. Accordingly I obtained Gärtner serum, paratyphoid B serum and paratyphoid A (Brion and Kayser) serum of known limits of agglutination with their homologous organisms, and tested the reactions of the bacillus I had isolated against these. I found that, while it was rapidly agglutinated by Gärtner serum in relatively high dilution, it was not agglutinated at all by paratyphoid B or A serum. It is also necessary here to observe that the identity of an unknown organism is not established simply because it reacts at some arbitrary dilution with a known serum, but it is the relative ultimate limits of agglutination of the unknown and known organism with homologous and heterologous sera which are of value. This leads to an important observation on serum diagnosis, which is of much interest to us as medical officers of health. Where Widal's reactions are done in large numbers in a laboratory, some arbitrary dilution is fixed on as a standard—1 in 50 in sixty minutes is a good average standard. This, with or without the clinical symptoms, is often accepted as complete proof of the existence of typhoid fever. But Boycott, in a remarkable paper which appeared in the *Journal of Hygiene* for 1906, clearly showed that sera may contain, besides the homologous or primary agglutinin, heterologous or secondary agglutinins, and that a number of cases diagnosed by such arbitrary standards as I have indicated are not typhoid fever at all, but paratyphoid fever, in the majority of instances caused by the *Bacillus paratyphoid B*, which I have already mentioned as belonging to Sub-group 3 in the classification I have given. The occurrence of such cases in Germany and America is estimated at 10 per cent. of those clinically called typhoid. Boycott gives the figures for England 3 per cent., but admits that this is probably too low. So that you see such cases occur in numbers sufficiently large

to show that they are more than mere matters of bacteriological interest; and especially are they important to the patient, as while the case mortality of paratyphoid is something like 1 to 3 per cent. at the most, that of typhoid is about 18 per cent., and it makes everyone happier all round when the prognosis is favourable. I have briefly indicated the groups of meat-poisoning bacilli and their methods of identification. The next point with which we are concerned is the distribution in nature of these various organisms.

For information on this subject we are mainly indebted to Morgan, who has isolated the *Bacillus enteritidis* of Gärtner and *paratyphoid A* from the fæces and scrapings of the intestines of various animals—viz., the rabbit, the guinea-pig, the sheep, the pig, the bullock, the calf and the horse. He has also shown that of these two organisms the *Bacillus enteritidis* of Gärtner has by far the greater virulence, for while he was unable to isolate it at all from 133 colonies by plating directly, he isolated the *Bacillus paratyphosus A* by plating directly in twenty-six out of 133 colonies; on the other hand, after passage through an animal, he found the *Bacillus enteritidis* of Gärtner twenty-one times, as against ten for the *Bacillus paratyphosus A*. Morgan also found the *Bacillus enteritidis* of Gärtner in the small intestine of a child, while Ford records several cases in which he found the *Bacillus enteritidis* of Gärtner in the large intestine (more especially the cæcum) in the human subject.

Savage, working at the same question in 1904-5, specially examined the excreta of two cows, one horse and one pig, and scrapings of the intestines of four cows and eight pigs, and found in four cases, all pigs, members of the Gärtner group, and in one of these they were present in great numbers.

This account of the distribution of the meat poisoning bacilli, is interesting, as it at once opens up the possibility of meat becoming infected after slaughter, or even after cooking. The frequency, too, with which these meat poisoning bacilli have been found in the pig's intestine may also account for the greater number of meat poisoning outbreaks due to pig meat. Outbreaks in which the food became infected after slaughter have been frequently recorded; and the last, I believe, was that recorded by myself, already referred to. In this particular outbreak, due to eating brawn, no illness of any kind occurred amongst

those eating the fresh pork or the sausages sold by the same butcher on the same day from the same animals, and inquiries failed to show that there had been anything amiss with the pigs prior to their being killed. Only those who ate the brawn were ill; and as the slaughterhouse was a private one, and the same persons were engaged in slaughtering the pigs and preparing the brawn, it is not difficult to understand how this latter article of food became infected from the intestines of the slaughtered pigs close by. In these days of "bacilli carriers," and from the work of Morgan and Savage which I have already quoted, it is obvious that food animals may be slaughtered which have not suffered from any septic or enteritic process due to *Bacillus enteritidis* of Gärtner, and yet harbour this bacillus in their intestinal tracts to infect, if due care be not taken, the meat after slaughter, and so spread disease.

In the class of meat poisoning outbreaks—and many have been recorded—where the *Bacillus enteritidis* of Gärtner gains access to the food after, or during, cooking, it is difficult to see what prophylactic measures, so far as meat inspection is concerned, can be adopted. But it is clearly objectionable that meat should be prepared on the same premises where slaughtering of animals is done, and still more objectionable if the same persons are engaged in both pursuits. In spite of the most scrupulous cleanliness, infection of the food may occur, and I do not think such a combination of occupations is in the best interest of the public health. When we come again to the question of meat having been infected with the *Bacillus enteritidis* of Gärtner, prior to the slaughter of the animal, how far does meat inspection take us? The mere naked eye inspection after slaughter does not help us very much, for meat containing the bacillus may look distinctly good. What is important in these cases is the history before death; and if the animal has suffered from any disease—especially septic or enteritic—which has necessitated emergency slaughter, then the greatest suspicion should be at once aroused. Under these circumstances there are several tests we may apply to ascertain the soundness of the meat. By the serum reaction, either after death or during the life of the animal, we can ascertain freedom from infection by the meat poisoning bacilli. Another method is to make a culture from the depths of the meat. If bacterial growth occur

within eight to twelve hours when the cultivation is exposed to a temperature of 25° to 30° C., then the meat should be rejected. According to Van Ermengem, this test is reliable eight days after the slaughter of the animal, and even though the meat already shows signs of putrefaction.

And just a word in regard to some practical points in case of an outbreak. In the first place, there is no evidence that personal contagion plays any part in the spread of an outbreak of meat poisoning. So the question of hospital isolation does not arise. The question of treatment does not greatly concern us as medical officers of health, and I am not aware that any anti-Gärtner serum has ever been prepared or used. But what we are concerned with is the cause. A very brief investigation will soon point to some article of food as the general cause. A sample of the suspected article should be secured, and that with the greatest possible promptitude. By anyone who has conducted a bacteriological examination of such material, which has become stale, the importance of this advice cannot fail to be appreciated. The *Bacillus enteritidis* of Gärtner in any food stuff is rapidly outgrown, if not killed, by the *Bacillus coli communis* and *proteus* and other organisms of intestinal type with which it is almost invariably associated. So every hour, every minute, is of importance in securing the sample and packing it in ice until the bacteriological examination can be begun, and this should not be delayed. Should any one or more of the sufferers succumb, then permission for a bacteriological examination of the various organs should be obtained without delay. Specimens should be collected from the spleen, heart-blood, liver and kidney with all possible sterile precautions and all possible haste. These should be packed in ice, again with sterile precautions, and the examination begun with the least possible delay.

In a paper of this kind it is not possible to give a detailed account of the methods to be used in a complete examination of the incriminated food stuff. But with reference to the bacteriological examination, I may say at once that what experience I have had has shown me that the primary culture medium should be a dulcete broth as recommended by Boycott. The majority of, if not, indeed, all, food poisoning organisms are dulcete fermenters; and by means of primary differential culture in dulcete media, the dulcete fermenters are

found in increased proportion. Next an animal experiment, or plating on Drigalski medium and selecting the blue colonies for further cultural tests, may be resorted to. Finally, agglutination reactions with known sera and the sera of persons affected should be done to complete the identification of the organism and its causal relationship with the outbreak. Besides a bacteriological examination, chemical tests for ptomaines and toxins should be performed, and so would the investigation be made complete.

REFERENCES.

- Bollinger (1876). Address delivered at the Fourth Session of the German Society for Public Hygiene in Düsseldorf.
- Bollinger (1880). Address delivered before the Medical Society of Munich.
- Boycott, A. E. (1906). Observations on the bacteriology of paratyphoid fever, and on the reactions of typhoid and paratyphoid sera. *Journal of Hygiene*, vol. vi, p. 33.
- Brieger (1885-6). Ueber Ptomaine (1885-6), part iii, p. 65.
- Buchan, G. F. (1907). An outbreak of food poisoning due to eating brawn. *Lancet*, vol. ii, 1907, p. 1604.
- Cathcart, E. P. (1906). The toxin of the *Bacillus enteritidis* of Gärtner. *Journal of Hygiene*, vol. vi, p. 112.
- Durham, H. E. (1898). On the serum diagnosis of typhoid fever, with special reference to the bacillus of Gärtner and its allies. *Lancet*, 1898, vol. i, p. 154.
- Durham, H. E. (1898). An address on the present knowledge of outbreaks due to meat poisoning. *British Medical Journal*, 1898, vol. ii, p. 1797.
- Van Ermengem (1897). *Zeitschr. f. Hyg.*, 1897, vol. xxvi, p. 1-21.
- Gärtner (1888). *Breslauer Aerztlich. Zeit.*, 1888, Nos. 21 and 22.
- Fischer (1893). *Deut. Med. Woch.*, 1893, p. 575.
- Ford (1904). Studies from the Rochfeller Institute, vol. ii, 1904.
- Morgan, H. de R. (1905). Micro-organisms of meat poisoning. *British Medical Journal*, 1905, vol. i, p. 1257.
- Ostertag (1904). Handbook of Meat Inspection (Wilcox).
- Savage, W. G. (1906). The Bacterial Examination of Water Supplies, p. 72.
- Smith, Theobald (1893). Bureau of Animal Industry. Bull. No. 6, Washington, 1893.
- Thresh and Porter (1906). Preservatives in Food and Food Examination, pp. 303-327.

SIXTEENTH INTERNATIONAL MEDICAL CONGRESS.—The 16th International Medical Congress will be held at Budapest in 1909—from August 29th to the 4th September—under the Patronage of His Imperial and Apostolic Royal Majesty, Francis Joseph I. Full particulars may be obtained from the General Secretary, Dr. E. Grösz, Budapest.