solution. Depending on the size of the dog, 125 to 175 c.c. of corpuscle suspension were then slowly injected into the vein toward the heart. While the transfusion was in process about 100 c.c. of blood were allowed to flow from an incision in the vein peripheral to the point of injection. It was necessary to work rapidly. If the heart again ceased to contract before the transfusion was begun, it was usually impossible to effect a return of rhythmical contractions even though intrapericardial epinephrin injection and cardiac massage were once more resorted to. In only two instances out of a total of seven dogs did the continuance of the injection of the corpuscles plus epinephrin start the cardiac contraction. Of the five dogs which were not resuscitated by the transfusion after their heart contractions had ceased a second time, it is interesting to note that at immediate necropsy in two cases the right ventricle and auricle were found distended with clots of blood which were distinctly darker than the fluid blood also present.

Fifteen dogs in which the transfusion was begun before the heart had again ceased to pulsate lived. Five control dogs, in which it was possible to stimulate a return of cardiac and respiratory activity as above, in which, however, no transfusions were performed, died within two to four minutes following the temporary resuscitation. Four control dogs in which 125 c.c. of Ringer's dextrose solution plus epinephrin were injected intravenously instead of the corpuscle suspension, died within from three to five minutes after the intravenous injection, despite the fact that cardiac contractions had again been established, as had also a return of the respiration.

Of the fifteen dogs that lived following the erythrocyte transfusion, two failed to regain consciousness. One died after thirty-six hours, of coma. It is interesting to note that at necropsy the right heart was found distended by a bright cherry red fluid blood in which were many small, dark or black, irregularly-formed coagula. The branches of the pulmonary artery contained here and there fragments of such coagula, though there were no gross evidences of lung infarction. The second dog which failed to regain consciousness lived twelve hours. This dog was ill before experimented on, but was used because of a scarcity of available dogs at that time. At necropsy a severe purulent rhinitis and purulent bronchitis and lobular pneumonia were found. The remaining thirteen dogs recovered consciousness usually before the vein had been tied or the skin suture completed. They either lay quietly or staggered about unsteadily. On the following morning they were apparently normal except for local reactions about the neck wound. One of the thirteen dogs appeared abnormal mentally immediately following the experiment. Following his transfusion and removal from the table he was able to run about quite vigorously. However, he snapped and bit at inanimate objects, ate his feces, and four days following his transfusion killed and partially disemboweled one of the other transfused dogs in his cage. He had to be killed before he could be removed from the cage. I regret to say that his brain was not examined. Five of the transfused dogs were killed seven to ten days after their transfusion because of a severe cervical cellulitis, due probably in part to imperfect surgical asepsis at the time of transfusion. Aside from their local infection, the dogs apparently had recovered from the experimental asphyxia. The six remaining dogs showed no after-effects either direct or indirect.

From the foregoing it will be noted that of the fifteen dogs transfused, twelve recovered with apparently no ill after-effects that could be attributed to the asphyxia. Two of the fifteen transfused dogs died without regaining consciousness. One of these was probably fatally ill before the experiment. From necropsy findings of the second it might be suggested that death may have been due to agglutinins in his blood for the corpuscles with which he was transfused. The third dog developed symptoms quite typical of the manias sometimes seen in human beings following acute carbon

monoxid asphyxiation. The possibility of an incidental rabies in this animal cannot, however, be ruled out, because the animal was disposed of before the brain could be examined.

The erythrocytes used in these transfusions had been preserved from three to seventeen days. Two dogs were transfused with corpuscles obtained seventeen days earlier, one dog with sixteen-day corpuscles, three with fourteen-day corpuscles, four with six-day corpuscles, and five with three-day corpuscles. The two dogs which died without regaining consciousness and the one which developed maniacal symptoms had been transfused with three-day corpuscles. Observations to detect hemoglobinuria, temperature disturbances, changes in the microscopic blood picture, etc., were not made. All of the control animals died.

CONCLUSIONS

Erythrocytes of some animals can be preserved intact over extended periods of time.

These corpuscles can again assume their physiologic function in another animal of the same species, and can successfully prevent death in carbon monoxid asphyxiation of this second animal, in about 75 per cent. of all cases in which indirect transfusion is begun while cardiac contractions can still be elicited.

The establishment of emergency stations where human erythrocytes could be obtained, just as pulmotor stations are now being maintained, should prove of invaluable service in saving the lives of human beings the victims of illuminating gas asphyxiation.

FOOD POISONING BY THE BACILLUS PARATYPHOSUS B

AN EPIDEMIC DUE TO THE ORGANISM ISOLATED
FROM PIE

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During July, 1915, an epidemic of food poisoning occurred at Westerly, R. I., and in an adjacent township of Connecticut. The epidemic was characterized by symptoms of an acute gastro-enteritis. The symptoms followed the eating of pie, which was obtained at a restaurant in Westerly. It is estimated that sixty persons were made seriously ill, four of whom died.

At the inquiry, many witnesses testified to having eaten pie, purchased from the restaurant in question, and yet remained unaffected. It was, nevertheless, firmly established that all those who were affected traced their symptoms to the pie. The owner of the restaurant stated that the dough was mixed Friday, July 2, and that a batch of pies was made on the following day for Sunday and Monday. This batch consisted of coconut-custard, custard, squash, lemon, chocolate, berry and apple pies. The preparation of these food products differed in no way from that employed for many years previously.

The symptoms appeared from twelve to nineteen hours after eating. In some cases the symptoms appeared as early as four and a half hours. They were characterized by epigastric pain, a burning sensation

from fauces to stomach, extreme thirst, and spasmodic contractions of the calves of the legs. Vomiting was a constant symptom and was of about six days' duration. The vomitus was green. Diarrhea was also present and was associated with tetanus. The stools were of a pea-soup consistency, greenish, and extremely foul smelling. The green color of the stools persisted for many days. The number of the dejections varied from ten to twenty daily. The temperature was elevated, reaching 103 in some cases. The pulse was normal. Two of the patients had a herpetic eruption around the mouth, and in one other a generalized erythema appeared for twenty-four hours. One child of 8 was rendered unconscious for two or three days, and presented a marked rigidity of the neck. In one of the fatal cases, there was a suppression of urine. Most striking of all was the mental depression. Convalescence was protracted.

Two men and one woman died within four days after the onset of symptoms. The fourth person, a man, died three weeks later. The persons who died were all beyond middle age. The first fatal case was that of a man aged 49. Symptoms of marked severity came on seven hours after eating and proved fatal in thirty-six hours. Two other members of the family, who had eaten pie at the evening dinner, became ill, whereas four other members, who had not had any pie, were unaffected.

There was no unusual taste or odor to the pies to excite suspicion; and the symptoms followed the eating of all the different kinds of pie of which this particular batch consisted. The gradual increasing number of cases of poisoning gave rise to much alarm. The State Pure Food and Drug Commission united with the officials of the State of Connecticut to determine the cause of the epidemic. Sanitary surveys were made; samples of pies and materials used in their preparation were submitted for chemical analysis. Prof. Clifford B. Morrison of the Connecticut Experiment Station reported the absence of arsenic or heavy metals and of poisonous alkaloids or other organic compounds. A similar report was made by Prof. Franklin N. Strickland of Providence. Samples were also submitted to our laboratory for examination. It is noteworthy that the laboratory investigations were conducted after the acute stage of the epidemic had passed. No field work, which might have enhanced the value of this epidemiologic study, was rendered possible.

The following samples were received in half-pint jars, partially filled: (1) milk; (2) dough; (3) pie-shortening; (4) coconut-custard pie; and in addition there was a small quantity of (5) egg flour, contained in a paper package. The small amount of material precluded any attempt to isolate a ptomain by chemical means. Accordingly, the bacterial content of the specimens merited inquiry.

The milk had a sour odor; a moderate amount of curd was formed. Smears from centrifugized portions showed a few cells; no streptococci were present. The bacterial count was 7,820,000 organisms per cubic centimeter. Smears were made direct from each of the four remaining specimens; bouillon, plain-agar slants, dextrose-agar stab cultures, and plates of plain-agar, dextrose-agar, and Endo medium were inoculated. Anaerobic cultures were also made; and some of the inoculated mediums were incubated at room temperature. One c.c. of each of the four bouillon cultures, containing the initial bacterial growth, was

injected into the peritoneal cavity of guinea-pigs. The two animals, inoculated with the growth obtained from the pie mixture and dough, died after eighteen hours. Postmortem examination revealed marked edema of the abdominal wall and a diffuse peritonitis. Cultures from the heart's blood showed a mixed infection in which the *Bacillus coli communior* and streptococcus were present. The guinea-pigs inoculated with the bouillon cultures, derived from the pie shortening and egg flour, survived.

The twenty-four hours' growth on the Endo plates, which were seeded with an emulsion of the pie, contained a mixture of red and of transparent, colorless colonies. The latter were transplanted on slant-agar. They yielded a profuse growth of gram-negative bacilli. These bacilli in subsequent cultures showed a slight tendency to pleomorphism. They possessed motility and showed the following cultural characteristics: Litmus milk at first became slightly acid, the acidity giving way to a permanent alkalinity. Gelatin was not liquefied, but gas was produced therein. With Hiss carbohydrates serum-water mediums, dextrose, lactose, galactose, mannite and maltose showed acid and gas formation; lactose, saccharose, dextrin and salicin were unchanged.

An eighteen-hour bouillon culture of the organism, thus isolated from the pie mixture, and termed for convenience "Strain 4," was employed in the determination of its pathogenicity. Three animals were each inoculated with 1 c.c.: one rabbit subcutaneously, one guinea-pig subcutaneously, and one other guinea-pig intraperitoneally. The two guinea-pigs died over night. A diffuse peritonitis was present in each animal. The organism recovered from the heart's blood and peritoneal cavity possessed the same cultural characteristics as the one injected. It was a gram-negative, motile coccobacillus, producing colorless transparent colonies on Endo plates and a permanent alkalinity in litmus milk. The rabbit died seventy-two hours after injection. Cultures taken from the heart's blood and peritoneal cavity, twenty-four hours postmortem, were in turn transplanted on Endo plates. Colorless colonies were obtained identical in character with Strain 4.

Feeding experiments were also performed. A kitten was fed with portions of the pie and suffered no ill effects. Another kitten was fed on sterile milk, which had previously been inoculated with Strain 4. A mild enteritis resulted.

Through the courtesy of Dr. A. S. Briggs of Ashaway, a specimen of blood was received from a patient, aged 20, who "had quite severe symptoms after eating pie." The blood was collected twenty-four days after the onset of symptoms. It is particularly noteworthy that this patient gives no history of any illness of over two or three days' duration prior to the attack of food poisoning. The blood serum from this patient agglutinated the typhoid bacillus, the paratyphoid bacillus B and Strain 4 in the dilutions of 1:10, 1:40, 1:80, 1:100 and 1:200 (the latter dilution showed partial clumping). The paratyphoid bacillus A was agglutinated only in dilution of 1:10. The patient's serum also agglutinated the organism recovered from the hearts' blood of the guinea-pigs and of the rabbit which had been inoculated with Strain 4. Moreover, Strain 4 was agglutinated by an antityphoid serum.

Thanks to Dr. G. T. Swarts, ten dried specimens of blood were received from individuals who similarly had suffered ill effects from eating the pie in question. The

specimens were collected one month after the onset of symptoms. Five of the specimens caused complete agglutination of Strain 4, three produced partial clumping, and two were negative. The question whether the organism isolated was capable of producing a soluble toxin invited solution. Accordingly a forty-eight-hour bouillon culture of Strain 4 was filtered through a Berkefeld filter. Two guinea-pigs and one rabbit were each inoculated with 1 c.c. of the bacteria-free filtrate. The animals developed no symptoms. Two animals were used as control, one guinea-pig was injected with sterile bouillon, and one other guinea-pig with the bouillon containing the growth of Strain 4. The latter died over night. All other observations were similarly controlled and repeated. In each instance, the observations found corroboration.

It is to be recalled that of the mixed cultures made from the ingredients of the pie, the bouillon inoculated with the dough proved fatal to a guinea-pig, as did the mixed culture from pie. The animals died either from the toxins in the food products or from the organisms present. It may well be assumed that Strain 4, if present, may have been overgrown by the streptococcus and the *Bacillus coli communior*. Since Strain 4 has been isolated only from the pie, the presumptive evidence is that the dough, also, contained the organism.

Thus the organism isolated from the pie mixture possesses the morphologic, cultural, pathogenic and immunologic characteristics of the *Salmonella* group. This group is composed of the *Bacillus paratyphosus* B, the *Bacillus suipestifer* and the *Bacillus enteritidis* Gaertner. It is noteworthy that the German school regards the *Bacillus paratyphosus* B, and the *Bacillus suipestifer* as identical species, applying the former designation if derived from human sources, and the latter if derived from animal sources. The English school, however, by means of agglutination and absorption tests, distinguishes between the two organisms. These tests of necessity were applied to Strain 4, as there was no opportunity forthcoming to determine the source of the food infection.

Rabbits were accordingly immunized against Strain 4, the *Bacillus paratyphosus* B, and the *Bacillus suipestifer*. A culture of *Bacillus suipestifer* was obtained through the courtesy of the American Museum of Natural History, New York. Three intravenous injections of a twenty-hour bouillon culture of each of the three organisms were administered to rabbits at fifteen-day intervals. The animals were bled approximately one week after the last inoculation. The first injection consisted of 0.001 c.c. of the living culture, the remaining two of 0.01 c.c. Rabbits injected with Strain 4 succumbed shortly before the second injection became due. The organism was recovered from the heart's blood and stools. Fortunately it was possible to obtain blood from one rabbit after it had survived a second injection of Strain 4. At that time the animal had a paralysis of the lower limbs and of the urinary bladder. This may account for the relatively low titer in the agglutinating properties of the serum.

The accompanying table indicates the agglutinating powers of each serum against Strain 4, the *Bacillus paratyphosus* B and the *Bacillus suipestifer*; also the effect on the specific agglutinin and co-agglutinins of each serum after saturation with each one of the three organisms in question. The similarity of the serum reactions, thus made evident, between Strain 4 and the *Bacillus paratyphosus* B establishes definitely the iden-

tity of the former. The *Bacillus enteritidis* Gaertner was not agglutinated by any one of the immune serums.

It will be noted, however (under heading 2), that Strain 4 does not remove all the agglutinins for the *Bacillus paratyphosus* B from a paratyphoid B serum. Repetition of the test yielded constant results. A similar anomalous reaction is recorded by Hutchens and Tulloch¹ in a strain of the *Bacillus paratyphosus* B isolated from yeast in a brewery. This yeast had been used as food for farmyard animals and produced symptoms of an acute enteritis.

The agencies whereby the *Bacillus paratyphosus* B gained access to the pie must of necessity remain a matter of conjecture. The cases of poisoning were limited to persons who purchased the food-product at this Westerly restaurant. There had been no change in the personnel of the restaurant for several months, which makes the restriction of the epidemic in point of time difficult to explain. If we accept the view of Bainbridge, the infection originated from a human source, possibly a paratyphoid carrier.

The distribution of the *Bacillus paratyphosus* B in nature has given rise to much discussion. In Germany, bacilli of the paratyphoid group have been isolated

TABLE OF ABSORPTION TESTS

Serum	Agglutination Limits for		
	Strain 4	B. Paratyphosus B	B. Suipestifer
1. Strain 4, original titer.....	1,600	800	100
Absorbed with B. suipestifer....	1,600	400	<100
Absorbed with B. paratyphosus B	<100	<100	<100
Absorbed with Strain 4.....	<100	<100	<100
2. Bacillus paratyphosus B, original titer.....	3,200	6,400	1,600
Absorbed with B. suipestifer....	800	6,400	<100
Absorbed with B. paratyphosus B	<100	<100	<100
Absorbed with Strain 4.....	<100	800	<100
3. Bacillus suipestifer, original titer.....	1,600	800	6,400
Absorbed with B. suipestifer....	<100	<100	<100
Absorbed with B. paratyphosus B	<100	<100	6,400
Absorbed with Strain 4.....	<100	<100	6,400

from milk by Fischer (1903), from water by Conradi (1904) and by Priefer and Keyser (1902), from sausage by Hubener (1908) and by Rommeler (1909), and from goose by Sobernheim (1911). Moreover, Uhlenhuth (1908) and Seiffert (1909) have obtained the organism from the intestinal contents of healthy pigs.² It was noted by Conradi that food containing these organisms could be eaten by human beings without the production of untoward symptoms. Morgan, Savage and Bainbridge, however, have obtained negative results in the examination of pigs' feces.

The prevalence of the organism in man has been the subject of much investigation. Conradi found the bacilli present in twenty-nine cases out of 250 typhoid convalescents, in patients suffering from tuberculosis and other diseases, and in healthy individuals. Prigge and Sachs-Muke³ examined the stools of 5,252 individuals and found the paratyphoid bacilli present in 108 cases, only thirty-eight of which showed symptoms of paratyphoid infection. Hubener and Viereck, in an examination of the stools of 4,000 soldiers, encountered the organism thirteen times in individuals who never

1. Hutchens, H. J., and Tulloch, W. J.: An Anomalous Strain of *B. Paratyphosus* (B), Jour. Path. and Bacteriol., 1913-1914, xviii, 431.
2. Quoted from Bainbridge, F. A., and O'Brien, R. A.: On the Paratyphoid Group of Bacilli, Jour. Hyg., 1911, xi, 68.
3. Prigge and Sachs-Muke: Paratyphusbazillenausscheidung bei Kranken und Gesunden, Klin. Jahrb., 1909-1910, xxii, 237.

presented signs of paratyphoid fever. Hubener recovered the organism twice in seventy-five examinations of the nasopharynx.⁴ Negative findings, however, were obtained by Sobernheim, who searched for the *Bacillus paratyphosus* B in 1,000 samples of feces of normal individuals, by Seiffert who examined 600 specimens of feces, and by Morgan in a study of 303 stools from children.⁵

Bainbridge and O'Brien, in criticism of the findings of German investigators, have submitted the organisms which the latter isolated from varied sources to absorption tests. As a result, the organisms fall into two groups. The first group, the *Bacillus suispestifer*, consists of those strains which were derived from food or food-poisoning cases. The second group, the *Bacillus paratyphosus* B, was obtained from cases of paratyphoid fever or chronic carriers. Bainbridge and O'Brien are disposed to regard "the human alimentary canal (including the bile passages) as the normal habitat of *Bacillus paratyphosus* B, while admitting that paratyphoid 'carriers' may cause infection not only directly but also by contaminating sound food, just as is the case with typhoid carriers. Under these circumstances, it is to be expected that the bacilli may occasionally be found in food, polluted by a carrier, although they have not met with such an instance."² Moreover, they are convinced that the presence of the *Bacillus paratyphosus* B in healthy human beings is unknown in England. In this connection, the findings of Bonhoff are of unusual interest. In the bacteriologic examination of the heart's blood in 6,476 cases at necropsy, he isolated the *Bacillus paratyphosus* B from twenty-nine cases. Five of the twenty-nine cases fall into a group which the author classifies as bacteremia as a coincident finding (*reine Bakteriämien als Nebentbefund*). This group comprised one case each of senility, pulmonary tuberculosis and chronic alcoholism, and two cases of inoperable carcinoma.⁶

The importance of carriers of paratyphoid bacilli has been further emphasized by epidemics which owe their origin to them. Thus, Konrich⁷ described an epidemic of paratyphoid fever affecting forty-four children in a surgical ward. A young girl, recently admitted to undergo treatment for rachitis, was held responsible because of the presence of the organisms in her stools. A similar outbreak of paratyphoid (B) fever, verified bacteriologically, involved sixteen surgical cases at a hospital in Roanoke, Va.⁸ A colored boy who was employed as a helper in the kitchen was the probable source of the infection, "which was spread by finger conveyance from case to case in the wards." The first outbreak in England of an acute gastro-enteritis due to the *Bacillus paratyphosus* B is recorded by Bainbridge. The symptoms were identical with those described in the Westerly epidemic. Fourteen persons in four households became seriously ill. The organisms were isolated from two specimens of stools, and their identity determined by absorption tests. No food was held responsible, as the dates of onset varied. The exchange of visits between the households at the time

of the epidemic suggests strongly the intervention of a carrier.⁹

In addition to the infections by contact mentioned above, there are epidemics recorded in which food material proved to be the vehicle of infection, because of contamination by paratyphoid carriers. Sacquépée and Bellot describe an epidemic of paratyphoid fever affecting nineteen in a French army corps of 250. The cook in camp had recovered a short time previously from an attack of paratyphoid fever. His blood agglutinated the *Bacillus paratyphosus* B, which persisted in his stools.⁵ Similar epidemics of paratyphoid fever occurred in various Japanese camps on the island of Waiawa. A native who brought vegetables to these camps proved to have the bacilli in his stools, although he gave no history of any illness during the two previous years.¹⁰ Of forty-five individuals who were taken sick after drinking some light beer, thirty-three agglutinated the *Bacillus paratyphosus* B; and in the stools in eight cases, the organism was present. It is supposed that the brewer had infected the product, which was of domestic manufacture.¹¹ French pastry was productive of acute symptoms in thirteen cases. The baker of the pastry was a recent convalescent from an attack of paratyphoid fever, which he had contracted from his son. The organisms were found in the urine of the baker. The food itself was not available for bacteriologic examination.¹² A fatal case, that of a private in the German army, yielded the *Bacillus paratyphosus* B in cultures from the intestinal contents, urine and spleen. The clinical symptoms were most acute in character. At the same time, there were among the civil population twenty cases of food poisoning due to the ingestion of sausages. From the stools of one of the victims, the *Bacillus paratyphosus* B was isolated.¹³

It may not be amiss to cite two other epidemics of food poisoning in which the organisms were recovered from the food product. Fried eel was responsible for symptoms in a family of six. Paratyphoid bacilli were obtained from specimens of the eel. The organisms were more numerous, however, in the center of the fried portions.¹⁴ Most noteworthy of all is the food infection which occurred at Wrexham, England, in August, 1910. Many of the features of this epidemic was strikingly similar to ours at Westerly.¹⁵ One hundred and seven persons in fifty-six families became seriously ill as the result of eating pork pies. Of this number five died. The symptoms were those of an acute gastro-enteritis, associated with herpes and mental depression. The pork pies originated from the same bakeshop. The *Bacillus paratyphosus* B was found in two specimens of suspected pork pies, and agglutinins for the organism in five persons who had eaten the pie in question.¹⁶ The organism was also

9. Bainbridge, F. A.: An Outbreak of Gastro-Enteritis Due to *B. Paratyphoid* B. *Brit. Med. Jour.*, 1910, ii, 1515.

10. Sinclair, A. N.: An Epidemic of Paratyphoid Fever, Origination from a Carrier and Dissemination by Vegetables. *Am. Jour. Trop. Dis. and Prev. Med.*, 1914, i, 612.

11. Kling, C., and Petterson, A.: Verbreitung von Paratyphus und ähnlichen Darmkrankheiten durch Dummber, *Centralbl. f. Bakteriologie*, Part I, 1914, lxxiv, Orig., p. 467.

12. Prigge and Sachs-Muke: Beobachtungen bei zwei Nahrungsmittel verursachten Paratyphusepidemien. *Klin. Jahrb.*, 1909, xxi, 225.

13. Oesterlen, O.: Zur Kasuistik der Paratyphus B. *Deutsch. mil. ärztl. Ztschr.*, 1911, xl, 655.

14. Müller, R.: Fischfleischvergiftung durch Bakterien der Paratyphus-Enteritisgruppe. *München. med. Wchnschr.*, 1914, lix, 471.

15. Williams, D. L.: Food Infection at Wrexham. *Jour. Roy. Inst. Pub. Health, London*, 1910, xviii, 725.

16. Trommsdorff, R.; Rajchman, L., and Porter, Agnes E.: A Severe Outbreak of Food Infection Caused by a Paratyphoid Carrier. *Jour. Hyg.*, 1911, ii, 89.

4. Quoted from Job, M. E.: Les infections à paratyphus B: étude nosologique et épidémiologique. *Rev. de méd.*, 1913, xxxiii, 181.

5. Quoted from Bainbridge, F. A.: The Etiology and Epidemiology of Paratyphoid Fever and Food Poisoning. *Proc. Roy. Soc. Med.*, London, 1910-1911, iv, Epidemiology. Sec., p. 51.

6. Bonhoff, F.: Ueber Paratyphusbazillenbefunde an der Leiche. *Virchows Arch. f. path. Anat.*, 1914, ccxvi, 321.

7. Konrich: Eine Paratyphusepidemie in einem Krankenhause. *Klin. Jahrb.*, 1908, xix, 247.

8. Lumsden, L. L.; Freeman, A. W., and Foster, W. B.: Paratyphoid Fever: Report of an Outbreak in a Hospital at Roanoke, Va. *Pub. Health Rep.*, 1913, xxviii, 1041.

found in the stools of the head cook. She gave no history of ever passing through any typhoidlike illness, and to the best of her belief, she had never before caused a similar outbreak.

It must not be overlooked that food may continue to harbor active pathogenic organisms, even after subjection to cooking temperatures. Of course, the method of preparation and the physical character of the food determine the extent of penetration by heat. In the interesting experiments performed by Sawyer,¹⁷ it was shown that a pan of spaghetti baked for half an hour in an oven, the temperature of which ranged between 207 and 214 C., had a temperature of only 28 C. in the middle, and 48 C. near the bottom. The spaghetti had been inoculated, for the purpose of investigation, with a culture of typhoid bacilli. Living bacilli were recovered from a depth of 2½ inches.

SUMMARY

The purport of this communication is:

1. To record an outbreak of food poisoning due to the *Bacillus paratyphosus* B which affected sixty individuals, of whom four died.
2. To indicate that the vehicle of infection was pie, from which the organism was isolated.
3. To emphasize the necessity of protecting public food supplies from "disease carriers."

MULTIPLE METASTATIC SARCOMAS OF
THE LUNGS

WITH REPORT OF A CASE

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Sarcoma of the lung whether primary or secondary is very rarely found. So few cases come under the observation of any one individual that the study of sarcoma of the lung depends almost entirely on reported cases. The literature on the subject is very limited. The study of various records seems to give such different results that the complaint is general that too few cases are reported and those few not carefully enough.

The clinical aspects of metastasis secondary to malignant disease are of considerable interest and importance. When the existence of a primary tumor is obvious, or when such a tumor has been more or less recently removed, evidences of disease in other organs must necessarily excite alarm and arouse suspicions of a metastatic manifestation.

Such a relatively straightforward sequence of events, however, very rarely exists. The primary growth may have been overlooked or the nature of the tumor, removed by operation, may have been in doubt. Again the interval between the primary growth and the secondary development may be so considerable that suspicion is at rest.

The case reported here, fortunately, has been observed throughout the course of its malignant developments and the history is complete from the very

beginning of the primary growth to the fatal termination due to multiple metastatic sarcomas of the lungs.

S. S. Burt¹ in 1900 reviewed all cases of metastatic sarcomas of the lungs recorded in the *Johns Hopkins Reports, the Proceedings of the Pathological Society of Philadelphia* and the *Boston Medical and Surgical Journal*, and reports one himself. In all there are twenty cases. In only three are the metastases multiple and in two of these there is other involvement than just the seat of the primary tumor and the lungs. His own case is very similar to the one reported here, in that the primary growth involved an extremity, which was amputated because of it, and that there followed multiple metastases in the lungs, without any recurrence of the growth at the site of amputation or elsewhere than in the lungs.

F. R. Greenwood² in 1899 reports a case in which the same order of events prevails, with, however, this striking difference that in his case there was a general glandular involvement.

A. A. Stevens³ gives a brief but comprehensive review of data collected by different observers. He discusses both primary and secondary malignancies of the lungs and reports three cases which came under his own observation, one a primary sarcoma of the right lung, one an endothelioma of the right pleura with metastases in the right lung, the third a hypernephroma of the left kidney with secondary sarcoma of the left lung.

A case recently reported is one by T. M. Martin⁴ in which the findings in the lungs were secondary to sarcoma of the uterus.⁵ The following case was observed in the Cincinnati General Hospital:

History.—V. B., a widow, aged 54, was admitted to the West Medical Service, May 16, 1915. Her chief complaint was pain in the left lower chest, through and through the lung at the scapular level.

Family History.—Her father had died of pneumonia after a brief illness, her mother of old age. She was one of a family of eleven children, three of whom died in infancy; the others were living and well. Her husband had been shot through the lung in the Spanish-American War. He died in 1908 of pulmonary hemorrhage. There was absolutely no history of tuberculosis or malignancy.

Past History.—In early childhood the patient had had measles, chickenpox, whooping cough and mumps. At the age of 22 she was ill with a slight attack of peritonitis, from which she fully recovered. At the age of 44 she had an attack of pneumonia with pleurisy, which ran an acute course and went on to prompt and complete recovery.

In the summer of 1912 patient noticed a firm swelling which appeared over the upper part of the right tibia just below the knee. This lump gradually grew in size and in September, 1913, was opened to allow drainage. No pus was found. The incisions were allowed to heal and had almost closed when there was evidence of an active tissue proliferation with protrusion of the tumor substance at the site of the first openings. The diagnosis of sarcoma was made, and Dec. 19, 1913, the leg was taken off above the knee by Dr. W. D. Hamilton at Mount Carmel Hospital, Columbus, Ohio. Dr. Coon, pathologist to that hospital, reported the specimen to be oat-grain cell sarcoma.

Onset.—During the winter of 1914-1915 patient began to feel pain under the left shoulder blade and under the left

1. Burt, S. S.: *Philadelphia Med. Jour.*, 1900, vi, 545.

2. Greenwood, F. R.: *Birmingham Med. Rev.*, 1899, xlvii, 88.

3. Stevens, A. A.: *Med. and Surg. Rep. Episc. Hosp.*, 1913, i, 333.

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