A FATAL CASE OF GASTRO-ENTERITIS DUE TO BACILLUS AERTRYCKE *VEL* SUIPESTIFER.*

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IT is always interesting to come across a new strain of the food-poisoning group and be able to refer it to its proper category. In the present instance we were able to do this (contrary to the usual experience) by agglutination tests alone. Absorption gave us valuable confirmation of our results, but was not absolutely necessary.

P. H., a wheelwright, aged 24, was admitted to the Mater Misericordiae Hospital, Dublin, at 3.30 p.m. on September 15th, 1915. I am indebted to Dr. Coyne, house-surgeon, for the following notes: The man was in a state of utter collapse, almost unable to speak, though quite conscious. Face pale and drawn, eyes sunken and dull, expression listless, mouth dry, tongue swollen and coated with thick brown fur, decubitus dorsal. The extremities were cold and blue, skin dry, no rash, pulse flickering and almost uncountable, respirations 40, temperature 101.4° F. Shortly after admission he became very restless and delirious, and so remained until he gradually sank into coma about an hour before death, which ensued at 10 a.m. on the 16th—eighteen and a half hours after admission. Whilst in hospital he had no vomiting, the bowels did not move, and there appeared to be suppression of urine. The history obtainable from the relations was scanty and unsatisfactory.

Dr. Walter Healy, who attended the patient at his own home, told me that the illness began five or six days previous to admission with violent and almost continuous vomiting and purging, accompanied by severe abdominal pains. His friends thought it might have been due to a meal of mackerel partaken of some days previously at a fried-fish shop in the neighbourhood. This, however, is mere surmise, for inquiry failed to establish the existence of any similar case in the family or neighbourhood, and the source of infection is therefore quite uncertain.

Autopsy.

This was performed by Dr. Coyne, and some of the organs reserved for more minute examination by me. It is unfortunate that no cultures were taken at the time. When I examined the specimens next day I found the stomach rather small, mucosa extremely rugose and thickened, most intensely hyperaemic, the tinge varying from dark brown to dark purple. It contained a little blood with some mucus. The intestines were almost empty, dark in colour, showed intense active hyperaemia both without and within, mucosa thickened, injected, velvety. There were no ulcers and the lymphatic apparatus (Peyer's patches, solitary and mesenteric glands) was quite unaltered. Spleen slightly enlarged, hyperaemic. Kidneys presented no well-marked naked-eye change.

Bacteriology.

Material was taken from the duodenum, jejunum, ileum, caecum, and rectum, and removed to the laboratory for bacteriological examination. The medium used was that of Drigalski in Petri dishes, and next day there was no difficulty in detecting blue, transparent-looking, non-lactose fermenting colonies in moderate numbers on all the plates. In addition to ordinary *coli*, streptococcus colonies came up in marked abundance. The blue colonies proved to consist of actively motile bacilli of typhoid-like aspect. Suspended in the hanging drop and treated with highly diluted antiparatyphoid B serum from two different sources they showed marked agglutination. They were not clumped by typhoid, Gaertner, or paratyphoid A serum. Consequently, at first I regarded this organism as paratyphoid B, and in my preliminary communication[1] I so called it. I was the more inclined to this identification on account of the fact, of which I was aware, that Bainbridge and Dudfield had recorded[2] an outbreak of gastro-enteritis (15 cases, none fatal) due to infection with *paratyphosus* B. Moreover, in four of their cases there was a history of dried haddock having been consumed on the evening prior to the outbreak of symptoms and another of the patients had eaten some cold dried fish—circumstances that seemed to bring their observations into line with mine.

I shall call the strain isolated from this case "H." Its morphological and cultural characters were those of the Salmonella group. It was a short, plump, actively motile rod, Gram-negative, and provided with a rather small number of flagella (about 3 to 6). On the Drigalski medium its colonies were permanently blue and transparent, with a tendency to milky opalescence. On Endo they were colourlesss with pink centres. Gelatine was never liquefied. On the carbohydrate media they behaved as follows (in shake cultures in ½ per cent. agar tinted with litmus and containing 1 per cent. of the sugar):

TABLE I.—*Fermentation Reactions of Strain "H."*

Class of Carbohydrate.	Variety.	Result of Growth.
Monosaccharides	Dextrose	Acid + gas.
	Fructose	Acid + gas.
	Galactose	Acid + gas.
Disaccharides	Maltose	Acid + gas.
	Lactose	No change.
	Saccharose	No change.
Trisaccharide...	Raffinose	No change.
Polysaccharide	Inulin	No change.
3-Atomic alcohol	Glycerine	No change.
5-Atomic alcohols	Xylose	Acid + gas.
	Arabinose	Acid + gas.
	Rhamnose	Acid + gas.
6-Atomic alcohols	Dulcitol	Acid + gas.
	Mannitol	Acid + gas.
	Sorbitol	Acid + gas.

Indol was not formed, neutral red was moderately reduced, and a pellicle was often formed on broth. Litmus milk was at first slightly reddened, but afterwards became blue and remained so. The cultures were inodorous.

Virulence.—The only tests made hitherto have been with white mice.

Mouse A was inoculated under skin of back with 0.1 of broth culture 3 weeks old.
Mouse B received the same amount intraperitoneally.
Mice C and D were fed with breadcrumb soaked with the same culture.

Mouse A died in thirty hours. A bloody oedema at seat of inoculation was the only *post-mortem* appearance noted. The microbe was recovered from the spleen, heart's blood, gall bladder, and seat of inoculation. From the intestines only *coli* was obtained.

Mouse B was found dead on the second morning after inoculation. There was very little evidence of peritonitis, but the intestines were filled with bright yellow, semifluid, slimy material. The heart's blood, spleen, and peritoneal fluid yielded "H" in pure culture; the intestinal contents were found to be swarming with it + *coli* in lesser amount.

Mouse C was found dead on the second morning—stomach distended with whitish matter resembling breadcrumb "H" isolated from contents of stomach and intestines, including rectum. Heart blood sterile. One colony of "H" from the spleen.

Mouse D was also found dead on the second morning. "H" recovered from contents of rectum (only).

The culture used was already three weeks old (it had stood most of the time at room temperature). The bacilli were quite virulent, but the question as to the extent to which the infection was assisted by toxicity still remains open. I have not yet had time to undertake its solution. The effect on mice is exactly what one would have expected from a member of the Salmonella group (to which the several rat viruses and mouse typhoid belong).

The organism therefore belongs to the group of paratyphoid B, and the two food-poisoners *suipestifer* and "Gaertner." For this group the name "Salmonella" has been proposed by Lignières, after Salmon, the American bacteriologist who discovered the bacillus of swine fever or "hog cholera," which is identical with the *Aertrycke* bacillus. The name "Salmonella" is convenient for the group as it avoids tiresome circumlocution and connotes a group of organisms which are culturally identical, though capable of being distinguished by serological tests.

For the sake of clearness, and in view of the fact that, owing to the prevalence of typho-coli infections during war time, much importance now attaches to the exact identification of pathogenic strains, I will enumerate the members of the Salmonella group, and explain their synonymy. The group comprises:

(*a*) *Bacillus paratyphosus* B, first isolated by Schottmüller in 1900–1, from cases clinically indistinguishable from enteric, and termed paratyphoid B by Brion and Kayser shortly afterwards. (Another typhoid-like organism, "paratyphoid A," isolated and named at the same time as "B," is clearly distinguishable from "B" by *cultural* as well as by serological tests, and is therefore excluded from the Salmonella group.)

(*b*) *Bacillus enteritidis* (*Aertrycke*), isolated in 1898 by Durham from the victims of the Hatton outbreak of food

* Based on a paper which was read in the Section of Pathology, Royal Academy of Medicine in Ireland, January 21st, 1916, but publication of which had to be withheld owing to pressure of other work.

poisoning (185 cases, 1 death), and by de Nobele, a pupil of van Ermenghem's, from a similar outbreak at a village of that name in Belgium (one fatal case). This organism has been shown to be identical with the so-called bacillus of hog cholera or swine fever, isolated in America by Salmon and Theobald Smith (in 1885) from pigs suffering from that disease, of which it was at first regarded as the cause. It appears to be identical with *B. breslaviensis* of Kaensche and Flügge. The name *suipestifer* undoubtedly holds priority. But, inasmuch as the causal relation of the organism to the disease in the pig has been called in question, and for a time at least to a large extent abandoned, it seems to me better to call it by a name that expresses its undoubted effect in human pathology (producer of enteritis).

(*c*) *Bacillus enteritidis* (Gaertner). This organism was first isolated by Gaertner in 1888 from an outbreak of food poisoning at Frankenhausen (57 cases, 1 death).

These three organisms together make up the Salmonella group. *B. paratyphosus* B usually gives rise to a symptom-complex resembling mild typhoid. The two *enteritidis* bacilli, "*Aertrycke*" and "Gaertner" are responsible for the much more acute and dangerous symptom-complex known as food-poisoning or meat-poisoning (often erroneously termed "ptomaine poisoning"). *Aertrycke* appears to occur oftener than "Gaertner," but the latter would seem to be the more virulent. The outbreak of meat poisoning investigated by me at Limerick in the year 1909, with its 74 cases and 9 deaths (all occurring within twenty-four hours of the outbreak of symptoms), is, so far as I know, the most violent hitherto recorded, and it was due to the Gaertner bacillus.

It is true that *B. paratyphosus* B has been known to produce gastro-enteritis of food poisoning type. At least one such observation has been recorded (by Bainbridge and Dudfield).[2] But the general rule is, as above stated, that *B. paratyphosus* B produces paratyphoid fever, whereas *Aertrycke* and Gaertner produce food poisoning. The three organisms are, as above mentioned, culturally identical. Gaertner is readily distinguishable from *B. paratyphosus* B by agglutination methods. But it is not so easy to distinguish between *B. paratyphosus* B and *Aertrycke* by such methods, and the distinction, though clearly established by Boycott[3] as far back as 1906, and confirmed by Bainbridge and O'Brien,[4] appears to be denied or ignored by German writers (see article by Uhlenhuth and Hübener[5]), who apply the term "paratyphoid B" to all non-Gaertner strains of this group occurring in the human subject, whilst they reserve the term *suipestifer* for all such strains isolated from the pig.

The question now arises whether the strain under discussion is a true *paratyphosus* B or an *Aertrycke*. In order to answer it, recourse was had to agglutination tests, supplemented, in the case of certain serums, by the absorption method of Castellani. The tests were applied to three cultures at the same time, namely:

1. *Bacillus paratyphosus* B—a typical strain called "R," isolated by me some years ago for a fatal case of paratyphoid fever.*
2. *Suipestifer vel Aertrycke*—a strain kindly sent me some years ago from the Lister Institute by Dr. C. J. Martin, F.R.S., director.
3. "H," the strain now under discussion.

All agglutinations were done by the macroscopic method, the serum dilutions being made first with normal saline, and then brought to the volume of 1 c.cm. in small test-tubes, with a suitably diluted suspension of the living micro-organism. The suspensions were always made from twenty-four-hours-old agar cultures, emulsified in sterile saline, filtered and thinned down with saline till they reached the proper grade of opalescence—which has to be learnt by experience. The tubes containing the dilutions were incubated for two hours at 37° C., then taken out of the incubator and examined within an hour or two. Readings taken next morning were found not to differ materially from those taken shortly after removal from the incubator. At first I tried twenty-four-hours-old broth cultures and agar suspensions killed with formaline, but found that the fresh living agar suspensions gave the clearest readings.

* The strain furnished by me to the Lister Institute, and used by Bainbridge and O'Brien in their work, *loc. cit.*

Absorption was carried out by mixing one volume of the antiserum with nine volumes of thick, opaque, filtered suspension of the heterologous strain. The mixture was left in the incubator for two hours at 37°, and the precipitated bacilli completely got rid of by prolonged high-speed centrifugation. Dilutions similar to those of the same serum unabsorbed were then put up and the agglutination test carried out as before. Most of the antiserums used were obtained from the Lister Institute through Dr. Harriette Chick, to whom my warmest thanks are due for so kindly complying with my numerous requests. Two antiserums—namely, one for the new strain "H" and the other for paratyphoid B strain "R"—were made by me specially for this purpose. I will now give the results in a series of tables and briefly comment on them.

I. Anti-*Aertrycke* serum C 38. From the Lister Institute, stated titre 1 in 2,000. This serum was tested several times, some of it having been sent out undiluted from the Lister Institute whilst other lots were sent out diluted 1 in 20. The results obtained with all were practically accordant. The following is typical.

TABLE II.—*Anti-Aertrycke Serum C 38 (from Lister Institute).*

Dilution.	Aertrycke.	"H."	Paratyphoid B.
1 in 100	+++	+++	+++
1 in 500	+++	+++	+++
1 in 1,000	+++	+++	+++
1 in 5,000	+++	+++	0
1 in 10,000	+++	++	0
1 in 50,000	+	0	0

+++ means complete agglutination, the bacilli often forming one large flocculus; supernatant liquid clear.
++ means large flocculi, but supernatant liquid not quite clear.
+ means minute flocculi, just visible to naked eye; liquid turbid.
0 means no change as compared with normal serum control.

Here we find that agglutination alone distinctly places "H" with *Aertrycke* rather than with paratyphoid B.

II. The same serum was now absorbed with paratyphoid B and retested. Result:

TABLE III.—*Anti-Aertrycke Serum C 38 after Absorption with Paratyphoid B.*

Dilution.	Aertrycke.	"H."	Paratyphoid B.
1 in 100	+++	+++	+
1 in 500	+++	+++	0
1 in 1,000	+++	+++	0
1 in 5,000	++	++	0
1 in 10,000	+	+	0
1 in 50,000	0	0	0

From this we see that saturation with paratyphoid B has removed practically all its own group-agglutinins, whilst leaving those specific for *Aertrycke* in well-nigh undiminished amount. "H" is agglutinated to precisely the same degree as *Aertrycke* and is therefore identical with it.

III. If all artificial antiserums gave such clear results as the above, the distinction between *Aertrycke* and paratyphoid B would not have given rise to so much discussion. But this is far from being the case. *Aertrycke* serum C 42 from the Lister Institute (stated titre 1 in 32,000) failed to yield accordant results, though the test was repeated several times. The one outstanding feature of this serum was the fact that it invariably clumped paratyphoid B at a higher dilution than its own homologous organism. This is an experience that often falls to the lot of workers with the typho-coli and meningococcus groups.

IV. Another serum which gave unsatisfactory results was one made in this laboratory against strain "H." At first fairly powerful (titre 1 in 10,000), it was found after keeping six months in sealed tubes (without antiseptic) to have lost strength (titre 1 in 1,000) for the homologous organism, while still fairly strong (1 in 5,000) against paratyphoid B. Absorption with paratyphoid B robbed it of

its power of agglutinating all three micro-organisms at a dilution exceeding 1 in 100.

TABLE IV.—*Test with Anti-paratyphoid B Serum from Lister Institute (C 51; stated titre 1 in 8,000).*

Dilution.	Aertrycke.	"H."	Paratyphoid B.
1 in 100	?	?	+++
1 in 500	0	0	+++
1 in 1,000	0	0	+++
1 in 5,000	0	0	++

V. As will be seen, even in very low dilutions (1 in 100), this serum (Table IV) yielded only slight, doubtful traces of clumping with *Aertrycke* and "H," whilst as against its own strain it went to 5,000 and beyond. It is therefore highly specific. "H" is thus shown to be identical with *Aertrycke* and absorption tests are not needed.

TABLE V.—*Test with Anti-paratyphoid B Serum from Lister Institute (C 89; stated titre 1 in 8,000).*

Dilution.	Aertrycke.	"H."	Paratyphoid B.
1 in 250	+	0	+++
1 in 500	+	0	+++
1 in 1,000	+	0	+++
1 in 5,000	0	0	+++
1 in 10,000	0	0	++
1 in 50,000	0	0	0

VI. Here again there is no need for absorption. This serum is highly differential, and gives the required information by means of agglutination alone.

VII. I also tried an anti-paratyphoid B serum prepared here by immunizing a rabbit against strain "R."

TABLE VI.—*Anti-paratyphoid B Serum, Strain "R."*

	Unabsorbed.			Absorbed with Aertrycke.		
Dilution.	Aertrycke.	"H."	Para-typhoid B.	Aertrycke.	"H."	Para-typhoid B.
1 in 100 ...	+++	+	+++	0	0	+++
1 in 500 ...	++	+	+++	0	0	+++
1 in 1,000 ...	0	0	+++	0	0	+++
1 in 5,000 ...	0	0	+++	0	0	++
1 in 10,000 ...	0	0	+++	0	0	++

I combine in the one table (VI) the results obtained with this serum in the unabsorbed and absorbed condition. It will be seen that the serum is highly specific, and might serve to at once distinguish between paratyphoid B and *Aertrycke*. Absorption removes the very last trace of the non-specific agglutinins, while the specific ones persist in well-nigh full force. The diagnosis of strain "H" as an *Aertrycke* or *suipestifer* is thus placed beyond all doubt.

REFERENCES.
[1] BRITISH MEDICAL JOURNAL, 1915, vol. ii, p. 782. [2] *Journal of Hygiene*, vol. xi, 1911, p. 24. [3] Ibid., vol. vi, 1906, p. 40. [4] Ibid., vol. xi, 1911, p. 68. [5] *Kolle and Wassermann's Handbook*, second edition, vol. iii, p. 1005, et seq.

PERCHLORIDE OF MERCURY POISONING BY ABSORPTION FROM THE VAGINA.

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THE unusual mode of administration of the poison makes the following case of interest.

An unmarried woman, aged 27, who had been in the habit of using mercury perchloride tablets dissolved in water as a vaginal douche, inserted one tablet (hydrarg. perchlor. gr. 8.75 in each) into the vagina, apparently under the impression that it would serve the same purpose as when dissolved and used as a douche. This was done at bedtime. I first saw her when she called at my consulting-room at 10 o'clock the following morning, complaining of pain and swelling of the vulva and giving a frank statement of what she had done.

I ordered immediate free douching with warm water. I saw her again at 1 p.m., and found that severe cramping pains in the abdomen had set in, accompanied by diarrhoea, followed later by severe and persistent vomiting. It is interesting to note that on examination I found the vaginal mucous membrane practically unaffected, though the external genitals were congested and oedematous.

At 7 p.m. I saw the patient again, when the symptoms were worse. The diarrhoea, pain, and vomiting continued; nothing would remain in the stomach; egg water was given without success. Hypodermic doses of morphine, gr. ¼, from time to time had little effect.

The following day a considerable quantity of blood was being passed in the motions, together with small flakes in the vomit. The patient complained of thirst, and was very somnolent. There was suppression of urine. On the third day the gums were swollen, inflamed and spongy, and of a dark colour, while the breath was fetid. Diarrhoea, pain, and vomiting continued persistently.

By the fourth day the pulse, which had up till then continued strong, began to waver. The salivary glands were swollen, and there was considerable salivation. The symptoms continued during the fifth day, and on the sixth day the patient showed signs of collapse, and died in the evening.

Post-mortem Examination.

The case was reported to the authorities, and Professor Harvey Littlejohn performed a *post-mortem* examination. The following conditions were found:

Skin.—Generally slightly jaundiced.

Heart.—Muscle pale and flabby. Both ventricles dilated, with resulting valvular incompetence.

Lungs.—Oedematous and deeply congested.

Stomach.—Mucous membrane showed slight signs of irritation and a few small haemorrhages.

Intestines—The upper part of the small intestine showed swelling of the mucous membrane, injection, and a general catarrhal condition. The whole of the ileum was deeply livid in colour externally, while internally the mucous membrane was of a uniform brownish-green colour, swollen, and in a necrotic condition. Here and there there were tumefied areas, with extensive blood suffusions under the mucous membrane, which were undergoing gangrenous ulceration. The necrosis was more marked in the lower part of the ileum and caecum. The contents of the small intestine consisted of a dark-brown fluid, apparently altered blood and mucus. The large intestine presented similar appearances, with marked infiltration of the submucosa.

Liver.—Some cloudy swelling, but otherwise appeared normal.

Spleen.—Engorged and somewhat enlarged.

Kidneys.—Enlarged and soft in consistence. Cortex pale and swollen. The capsule stripped easily. The medulla showed areas of congestion.

Pancreas.—No obvious changes.

Uterus.—There was no enlargement. There was no peritonitis.

Microscopic Examination of Tissues.

Microscopic examination of the intestine, kidney, and liver was carried out at the Pathological Department of the University of Edinburgh by Mr. R. Muir. The following is his report:

Large and Small Intestine.—Sections show extreme and extensive necrosis of the mucosa. Large numbers of intestinal bacteria have found this necrotic surface a suitable medium for their growth, large masses of these being found. The submucosa is swollen and extensively infiltrated by fibrinous exudate, especially in the small intestine, with some areas of leucocytic infiltration. This coat also shows marked necrosis, recognized by the complete loss of the nuclear staining reaction. The muscular coats also show necrosis, the fibres showing a hyaline degeneration with loss of nuclei. There is some cellular infiltration of the peritoneal coat, but this is not a marked feature. There are also present throughout these coats deposits of pigment, probably derived from blood. These have no special relation to any particular tissue, and may be *post mortem*. The whole condition is intensely necrotic with fibrinous infiltration.

Kidney shows extensive necrosis and catarrh of the renal epithelium of all the secreting tubules, especially in the convoluted series, many of these degenerated epithelial cells showing very marked granular degeneration going on to calcification. This calcification is very irregular in distribution among the tubules; the granules vary in size and have a strong affinity for the basic stains. The collecting tubules do not show so much necrotic change, but they contain granular and hyaline casts. The connective tissue of the renal substance is not much altered and the capillaries in the tuft show some slight thickening of their walls, but no necrotic change. The condition of the kidney is mainly that of intense parenchymatous degeneration going on to calcification.

Liver on the whole shows no gross changes, in fact it is extremely well preserved tissue. There is some degree of congestion and also some slight pigmentary deposit in the hepatic cells which at parts show some slight fatty degeneration. A striking feature is the well-preserved appearance of the liver cells, with numbers of nuclei undergoing mitosis. It suggests