PARACHOLERA.

BY

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THIS term was introduced by me some years ago to denote cases which, though clinically hardly distinguishable from cholera, are due to vibrios somewhat different from true *V. cholerae* (Koch). As well known for many years, vibrios have been described which are somewhat different from Koch's germ, and Hewlett, Cunningham, and myself have suggested that the disease may be due to a group of closely allied vibrios. I will not consider in this paper the numerous cholera-like vibrios found in water, and will limit myself to supposed pathogenic vibrios found in the stools of cases presenting cholera-like symptoms. The most important of these vibrios are the following:

1. *Finkler and Prior's organism*, isolated from cases of so-called cholera nostras in Germany many years ago. Its pathogenicity has been doubted by several observers. According to certain authorities, it would appear that the original strain has been lost and that no modern serological tests have been carried out with it.

2. *Pasquale's Vibrio massahuac*, isolated from cases clinically indistinguishable from true cholera in Massowah

in 1895. According to Pasquale, the organism differs from *V. cholerae* (Koch) in having two terminal flagella at each end. Durham found that this vibrio was serologically identical with a vibrio isolated from the water of certain German rivers by Dumber.

3. *Ruffer's vibrios*. Ruffer has isolated certain vibrios from Mohammedan pilgrims returning from Mecca and suffering from various diseases, but with no cholera symptoms. He has compared these "El Tor vibrios" with vibrios he has isolated from other sources, and distinguishes four groups. Neufeld and Haendel, however, having examined some of these vibrios, consider that they are true cholera.

It must be noted, as observed by Grieg, myself, and others, that in cases of true cholera, together with true *V. cholerae*, vibrios may be isolated which are not agglutinated by cholera serum.

Vibrio kegallensis (Castellani, 1913).

This vibrio, for which I suggested also the name *V. paracholerae*, has been isolated by me, as stated in previous publications, from cases in Ceylon clinically scarcely distinguishable from cholera, with profuse serous diarrhoea, severe vomiting, muscular cramps, etc. It was very abundant in the stools and the only species of vibrio

TABLE I.—Showing the Principal Characters of Three Strains—Serologically Identical—of *V. kegallensis* and Seven Strains of Serologically True Cholera.

	<i>V. kegallensis</i> .			True Cholera Vibrios.						
	Strain 1.	Strain 2.	Strain 3.	No. 1 (Saigon).	No. 2 (Ceylon).	No. 3 (Ceylon).	No. 4 (Ceylon).	No. 5 (Ceylon).	No. 6 (Ceylon).	No. 7 (Ceylon).
Motility ...	+	+	+	+	+	+	+	+	+	+
Litmus milk ...	A Pt C	A s Pt C	A s Pt C	A s	A s	C	A	A C	A C	A
Lactose ...	A-K	A	A vs	A s	A s-K	A	A	A s	A s	A
Saccharose ...	A	A	A	A	A	0	A	A	A	0
Dulcitate ...	0	0	0	0	K	K	K	K	K	K
Mannite ...	A	A	A	A	A	A	A	A	A	A
Glucose ...	A	A	A	A	A	A	A	A	A	A
Maltose ...	A	A	A	A	A	A	A	A	A	A
Dextrine ...	A vs	A	A vs	A	A vs	A	A vs	A	0	0
Raffinose ...	0	0	K	0	0	K	K	K	K	K
Arabinose ...	A	A	A	0	K	A vs	0	K	A vs	A vs
Adonite ...	0	K	0	0	0	K	K	K	K	K
Inulin ...	0	K	0	0	0	K	K	K	K	K
Galactose ...	A	A	A vs	A	A	A	A	A	A	A
Sorbitol ...	0	K	0	0	0	K	K	K	K	K
Laevalose ...	A	A	A	A	A	A	A	A	A	A
Inosite ...	0	K	0	0	0	K	K	K	K	K
Salicin ...	A	K	0	0	0	K	K	K	K	K
Amygdalin ...	A	0	0	0	0	K	K	K	K	K
Isodulcitate ...	0	0	0	0	0	K	K	K	K	K
Erythritol ...	0	0	0	0	0	K	K	K	K	K
Glycerine ...	A	0	0	A vs	A vs	0	0	K	0	K
Indol ...	0	0	0†	+	+	+	+	+	+	+
Voges-Pro.	0	0	0	0	0	0	0	0	0	0
Red nitrates ...	0	0	0	+	0	+	+	+	+	+
Neutral red ...	0	0	0	0	0	0	0	0	0	0
Gram ...	0	0	0	0	0	0	0	0	0	0
Gelatine ...	+	+	+	+	+	+	+	+	+	+
Serum ...	+	+	+	+	+	+	+	+	+	+
Broth ...	T	G T P	G T P	G T P	G T P	G T P	G T P	G T P	G T P	G T P
Peptone water ...	G T	G T	G T†	G T P	G T P	G T P	G T P	G T P	G T P	G T P

* Or trace.

† Or P very slight.

ABBREVIATIONS.—A = Acid. C = Clot. K = Alkaline. A-K = Acid then alkaline. T = Turbidity. G T = General turbidity. P = Pellicle. Pt C = Upper portion peptonized and clot at the bottom (milk). s = Slight. vs = Very slight. 0 = Negative result—namely, neither acid nor clot in milk, neither acid nor gas in sugar media, non-production of indol, non-liquefaction of serum or gelatine as the case may be.

TABLE II.—Showing Agglutination Limits of Serums Derived from Hyperimmunized Rabbits.

Serums.	<i>V. kagallensis</i> Strain 1.	<i>V. kagallensis</i> Strain 2.	<i>V. kagallensis</i> Strain 3.	<i>V. cholerae</i> Strain 1.	<i>V. cholerae</i> Strain 2.	<i>V. cholerae</i> Strain 3.	<i>V. cholerae</i> Strain 4.	<i>V. cholerae</i> Strain 5.	<i>V. cholerae</i> Strain 6.	<i>V. cholerae</i> Strain 7.
<i>Kegallensis</i> Strain 1...	2,000	2,000	1,600	0	0	0	0	0	0	0
<i>Kegallensis</i> Strain 2...	2,000	2,000	2,000	0	0	0	0	0	0	0
<i>Kegallensis</i> Strain 3...	2,000	2,000	1,600	10	0	0	0	0	0	0
True cholera (Berne)	10	20	20	> 4,000	> 4,000	> 4,000	> 4,000	> 4,000	> 4,000	> 4,000

present. It was found also in the water of a well in Matale, near which a small epidemic of cholera-like cases occurred. The germ is microscopically and culturally very similar to *V. cholerae*. It is very motile, like true cholera, and it is easily stained by the usual dyes; it is Gram-negative. In stained preparations it has the same shape as the true cholera vibrio, but is perhaps slightly larger and thicker. On agar and gelatine and serum the growth is similar or identical to true cholera. Gelatine and serum are liquefied. Milk is generally rendered acid and clotted or peptonized. In peptone water the pellicle is very slight or absent. The germ does not produce indol, or only an extremely slight trace. It must be remembered, however, that certain strains of true cholera vibrio may produce only a very slight amount of indol, and may practically produce no pellicle in peptone water. The production of haemolysis is inconstant with all the strains of *V. kagallensis* I have examined.

Serological Reactions.

V. kagallensis is not agglutinated by true cholera serum in dilutions higher than 1 in 10 or 1 in 20; cholera vibrio is not agglutinated by *V. kagallensis* serum. The absorp-

tion test and Pfeiffer's test confirm the results obtained by the agglutination test.

Pathogenicity.

I have been unable to reproduce the disease in the lower animals by feeding experiments, but, as is well known, this is the case also with true cholera. Considering that the vibrio was present in the stools of the patients in enormous numbers, and was the only species of vibrio present, the probabilities are, in my opinion, that it is pathogenic.

CONCLUSIONS.

1. The term "paracholera," as I suggested some years ago, might be used to denote cases of cholera-like diseases due to vibrios, which are specifically different from *V. cholerae* (Koch), though closely allied to it.
2. In Ceylon cases of paracholera occur which in all probability are due to *V. kagallensis* vel *paracholerae*.

REFERENCES.

Castellani: *Journal Ceylon Branch, British Medical Association*, 1913 and 1914; *Reports to the Advisory Committee for the Tropical Diseases Research Fund for the Year 1914*, etc. Hewlett: *A Manual of Bacteriology*, 1914. Ruffer: *Bacterial Diagnosis of Cholera*, Alexandria, 1907.

A CASE OF CHRONIC NON-TUBERCULOUS PERITONITIS IN A CHILD.

BY

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As I am unable to find any record of a precisely similar condition to that which occurred in the following case, I venture to consider it sufficiently singular to justify its publication. As regards general appearance and all clinical manifestations, few would have hesitated to diagnose the complaint as the ascitic form of tuberculous peritonitis.

A boy, aged 4½ years; when first seen very bright and intelligent; had suffered from no previous illnesses excepting measles. As a baby he had been fed on cow's milk; his five brothers and sisters were all healthy and there was no tuberculosis in the family. The history of the beginning of his present complaint was indefinite. For about four months he had complained of pain in the "stomach" and occasionally vomited after food; there was no diarrhoea. The abdomen during this period had become larger and larger and loss of flesh had been considerable. There was no suggestion of injury.

Condition when First Seen.—The chest, face, and limbs were very emaciated; the abdomen was large and prominent. The heart and lungs appeared quite normal; pulse 90, respirations 23, temperature 98.8°. A large amount of ascitic fluid was present; there was no pain even upon firm pressure. The subcostal circumference was 24½ in.; the umbilical 21½.

Subsequent Progress.—The condition was diagnosed as tuberculous peritonitis; he was kept in bed and given syrup of iodide of iron; oleate of mercury ointment was rubbed into the abdomen daily. Two days after admission he was tested by von Pirquet's cutaneous reaction. On the right arm bovine tuberculin was used and on the left human tuberculin, with a control underneath each. Both proved positive, the intensity of the two reactions being about equal. The circumference of the abdomen in seven days had increased from 24½ in. to 25½ in. in the case of the subcostal circumference, and from 21½ in. to 22½ in. in that of the umbilical. Owing to the respiratory embarrassment—the respirations were now 40—five pints of dark brown fluid were removed by laparotomy; no glands were felt inside the abdomen, nor were any tubercles seen. A small piece of the parietal peritoneum, cut out and subsequently examined microscopically, showed no tubercles.

Ten days later the wound had quite healed and the stitches were removed, but there was still a small amount of fluid. During the time he had been under observation the temperature had varied between 97° and 98.8°; at no period had it been higher. At the end of another week the temperature suddenly went up to 100° and a purpuric rash appeared over the chest

and arms. A few hours later he became comatose and died the next morning.

The necropsy afforded no evidence of tuberculosis. In the abdomen was a fairly large quantity of fluid and numerous adhesions; the visceral and parietal peritoneum was enormously thickened. The omentum and mesentery were somewhat rolled up, and contained much inspissated pus between and around their layers. Pus of a similar nature was present on other parts of the peritoneum. No enlarged glands were found either in the abdomen, mediastinum, or neck; there was no sign of tuberculous ulceration of the intestine. The heart and lungs were normal, but the stomach wall was very thick. The spleen was soft and not enlarged; the liver indicated chronic congestion; the kidneys, brain, and meninges were normal. With such *post-mortem* revelations it can scarcely be maintained that the *Bacillus tuberculosis* was the organism responsible for the pathological changes, although clearly the condition was due to bacteria of low virulence.

Of the many varieties of chronic peritonitis, the following only need be considered in connexion with this case: (1) Diffuse adhesive peritonitis, (2) chronic proliferative peritonitis, (3) tuberculous, (4) gonorrhoeal, (5) pneumococcal, and (6) idiopathic (so-called) peritonitis.

Although undoubtedly the condition found at the necropsy could be described as diffuse adhesive peritonitis with suppuration, it does not agree with the type to which this name is applied, inasmuch as the latter follows an acute attack. The case differed from one of proliferative peritonitis in that pus and numerous adhesions were present and that the etiological factors were not the same.

The onset and course of the case were certainly more or less typical of chronic tuberculous peritonitis. There was a gradual subacute onset; the temperature varied between normal and subnormal. According to Hutchison, however, the ascitic effusion is rarely large; in this case it was sufficiently great to warrant laparotomy. Further, had the case been tuberculous, one would have found at least some *post-mortem* evidence of tuberculosis. In tuberculous peritonitis the effusion is usually straw-coloured, though occasionally it may be haemorrhagic; in this case it was dark brown. When ascites is present, the peritoneum is almost invariably found to be studded with tubercles.

It has been said that gonorrhoeal peritonitis has been found in males associated with a gonorrhoeal infection. In the case under discussion there is nothing to suggest this.