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*.

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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.

Pneumococcal peritonitis is more often primary or secondary to some other source of infection than a complication of lobar pneumonia. It is most common in female children, and is very acute; the pus is said to be thin, widely diffused, and with much fibrin. According to Still, primary peritonitis in children beyond the stage of infancy is nearly always tuberculous or pneumococcal. Records of 91 cases of pneumococcal peritonitis in children under 15 years of age, collected by Annand and Bowen, show that the symptoms were always severe, closely resembling, with one exception, the clinical course of diffuse perforative peritonitis. There was the same sudden onset, often with rigors and violent and persistent pain, frequent vomiting, advancing prostration, and early death. In children, however, this condition has been known to give rise to chronic encapsuled abscess, and it is conceivable that the chronic suppuration in this case may have been due to a strain of pneumococci of low toxicity.

Allchin, however, mentions certain forms of peritonitis attended with suppuration which run "a course the duration of which may justify their being designated as chronic." They may continue for many months, the severity of the local condition and the toxic manifestations being insufficient to cause death, though the general health of the sufferer steadily deteriorates. "Such cases," he says, "have been referred to as 'acute progressive fibrino purulent peritonitis.' It is only in the sense of their duration that they are to be regarded as chronic, being rather of the nature of a succession of acute or sub-acute attacks of localized character." Since this patient's illness did not commence with an acute attack, and during the course of observation showed no rise of temperature indicative of an acute exacerbation, it does not coincide with Allchin's description of acute progressive fibrino-purulent peritonitis.

It appears evident that the end was brought about by a sudden rise in the toxæmia, a fact which also accounts for the purpuric rash. As the evidence in favour of pneumococcal infection is not great, the most one can say is that the case was a somewhat anomalous type of chronic suppurative peritonitis and due to an organism of low virulence, such as *Bacillus coli communis*. Had the clinical appearance not been so typical of tuberculous peritonitis, one would have endeavoured to cultivate the organism from a swab taken at the time of operation. My acknowledgements are due to Dr. T. R. Whipham for his courtesy in permitting me to make use of the case.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

THE TREATMENT OF LAMBLIA INFECTIONS.

I was interested to read in the JOURNAL (March 18th, p. 407) of Drs. Woodcock and Penfold's experiences in the treatment of *Lambliæ* infections. In a paper I recently published in the *Journal of Tropical Medicine and Hygiene* I laid stress upon the importance of repeated and careful examinations of the stools both before and after treatment, and also mentioned some drugs tried by myself. In the doses in which I used them, emetine, beta-naphthol, and methylene blue all failed, completely to sterilize the patient of his infection. An apparent improvement occurred after the latter two, but prolonged examinations showed that this was only of a temporary nature.

I also described toxic symptoms following the administration of the methylene blue. Since then Major Marshall, of Edinburgh, has drawn my attention to a paper by himself and Gee published long ago in India, in which similar symptoms were met with after the use of this drug for malaria. He believes it is due to the difficulty of getting samples of the drug free from zinc chloride, and recommends giving it with extract of hyoscyamus to prevent bladder symptoms. Of other drugs I have used, bismuth alone, salicylic acid alone, salol, thymol, and cyllin have also proved ineffective.

It is specially interesting, then, to hear of Drs. Woodcock and Penfold's apparent cure of a case by the combined use of beta-naphthol and bismuth salicylate. Before finally

concluding, however, that this is a cure, further examinations should be carried out especially after the administration of good saline purges. Though very constant in certain cases, the passage of the cysts in others is equally inconstant, and they apparently may disappear for quite a long time of their own accord. An attack of diarrhoea or a purge often quickly brings them back again.

I recently saw a case heavily infected with *Trichomonas* and proposed treating him with methylene blue, but before doing so I thought I would observe him for a week. The trichomonads disappeared of their own accord, and so far (one month) have not again put in an appearance. If I had given the drug I would naturally have concluded that it had made them disappear. Turpentine, so lauded by French workers, and guaiacol carbonate I note also failed in some of the authors' cases. I am certain that many so-called successes, if followed up for a sufficiently long period (six months to a year), would have to be written off and debited to the failure column. Drs. Woodcock and Penfold's first case approaches the time limit given, though one would like to know how many times "several" really means. Their other cases, examined four and three days after treatment, of course give far too short a time to base any definite conclusions upon.

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A CASE OF POTASSIUM CHLORATE POISONING IN AN INFANT.

I FOUND a child aged 4½ months in a prostrate condition. There was marked pallor, with cyanosis of ears and lips and of the hands and feet, which were cold; breathing was quickened and shallow, but not jerky or noisy. The child lay still with eyes open, some contraction of the eyebrows, and an occasional peevish cry. The rectal temperature was normal; the pulse, 204, could be felt at the wrist. There was no cough, and nothing was found abnormal in the lungs. There was no vomiting and nothing abnormal about the abdomen. Some castor oil had been given before arrival, and the bowels had acted normally. The appearance suggested profound toxæmia. The diet had consisted of cow's milk, cream, and sugar of milk; the feeds had been prepared with scrupulous care, the mother herself being a medical graduate. Poisoning by gas-stove fumes was excluded, and the cream used was found on inquiry to be free from any preservative, such as boric acid.

It was noticed on the previous day that the feeds had been taken with some reluctance, but nothing abnormal was noticed about the child until the early morning of the day when first seen. When seen three hours later some brown-black material, the size of a small pea, found on the napkin was thought to be probably faecal until some greenish-yellow urine containing particles of similar material was seen issuing from the urethra. It was evident great destruction of the haemoglobin was in progress, and I suspected a chemical poison. Rough tests showed the supposed sugar of milk to be an inflammable salt of potassium, subsequently proved to be potassium chlorate. Death ensued without convulsions.

Post-mortem Examination.—The musculature was of a dusky red colour. The heart and blood vessels contained soft chocolate-coloured clot, and the walls of the aorta showed patches at intervals of brownish discoloration. The liver, spleen, and kidneys were chocolate in colour, with a "burnt sienna" coloured exudate on scraping. The calices of the kidney and the ureters and bladder were filled with a soft, friable brownish-black material which was not blood clot. The bladder also contained some turbid yellowish-brown fluid.

Reading.

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AT a meeting of the American Medical Library Association held at Washington in May, 1915, Mr. James F. Ballard urged that all medical libraries should adopt a uniform method of preparing statistics of their contents, and the following recommendations were made:—Everything of over 100 pages is a volume; everything of any size, if bound, is a volume; everything under 100 pages, if unbound, is a pamphlet. Duplicates should not be counted, except second copies of books in general use. Uncatalogued material and incomplete and unbound periodicals should be included.