

PARATYPHOID FEVER—A STUDY OF FATAL CASES

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With Plates 6-11

FATAL cases of paratyphoid fever are sufficiently uncommon to be worthy of record. The following account is based on two cases of paratyphoid A and fifteen cases of paratyphoid B with their post-mortem appearances.¹

Although in general paratyphoid is a reduced image of typhoid fever, the occurrence of severe and fatal cases shows that it has potentialities for greater mischief, and that under conditions favouring it the disease might break out in severe epidemic form—a consideration which bears upon the question of preventive inoculation.

The clinical picture of the severe case conforms so closely to that of typhoid that a complete description of the signs and symptoms would serve no purpose. A certain diagnosis between the two must be based on the laboratory findings.

The onset calls for no lengthened description. In only eleven of the cases could it be accurately noted. Abdominal pain and diarrhoea were the most frequent, occurring at onset in nine and eight cases respectively; together with vomiting they were the features of three cases which had a sudden onset. Head-ache occurred in six, cough in three, and epistaxis in two cases. The tongue resembled that found in typhoid.

In thirteen cases spots were present, and in some instances they were of larger size and less regular outline than those of true typhoid.

Abdominal distension is often noticeable by its absence, while in the toxic patients the 'typhoid state', with its wandering and low delirium, is less often a feature.

Respiratory System.

Rapid breathing is evidence, but not, like the pulse, a constant feature of a severe infection. In some cases it is not associated with definite signs in the lungs, and would appear to be the effect of the toxæmia on the central nervous system; a variability in the rate is suggestive of this type.

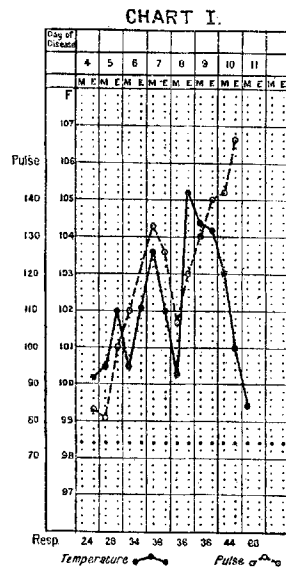
In four out of the seventeen cases the respiratory manifestations were so definite as to be a presenting feature of the clinical picture, and these cases are worth further consideration.

¹ Previously referred to in outline in *British Medical Journal*, Nov. 13, 1915, and *Proc. Roy. Soc. Med.*, Dec. 1915.

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Massive Pneumonia.—*Case 4, Chart I.* Developed the signs of lobar pneumonia at both bases (chiefly the right) on the sixth day of the disease; became delirious and died four days later, that is, on the eleventh day of the disease. At the autopsy the right lower lobe was solid and sank in water, and there was a little lymph on the pleurae and between the lobes. The left lower lobe was congested. There was early typhoid ulceration of the small intestine.

Was this pneumonia caused by paratyphoid or a supervening pneumococcal infection? It has been shown that lobar pneumonia can be caused by *Bac. typhosus* and paratyphosus alone, though probably more often the pneumococcus is in association. In this case there was an abrupt beginning to the lung invasion, as the record of the temperature, pulse, and respiration shows. There was no sputum, and no culture from the consolidated lung was made.



Case 3, Chart II. Massive pneumonia with cavity formation from necrosis. The respiration rate was high from the time of admission (eighth day) to hospital and the colour dusky. On the fifteenth day the resonance and breath sounds were impaired at both bases posteriorly; on the twenty-third day these signs were more marked, and as the 'spiked' temperature suggested the possibility of suppuration the chest was punctured, but without result. A small quantity of saline was then injected from a syringe into the lung, sucked back, and put into broth, but with a negative result. On the twenty-seventh day there were many crepitations heard over the lungs posteriorly; the breathing was about forty and increasingly embarrassed. On the twenty-ninth day the patient died. A comparison of the foregoing signs with the post-mortem findings brings out once more what inadequate information physical examination may afford of the actual condition of the lungs. We all knew from the patient's general condition that the lungs were more extensively involved than the physical signs denoted. At one time the possibility of concurrent tuberculosis came to mind, and the sputum was examined for tubercle bacilli twice with negative results.

The necropsy showed definite ulceration of the small intestine. Both lungs were almost completely consolidated, though the left was more affected than the right. Some thin lymph covered the apex of the upper lobe of the left lung. The anterior third of the upper lobe was aerated, though congested and oedematous. The posterior two-thirds, with the exception of a thin marginal strip, was solid. In this solid area the tissue was red-brown in colour and absolutely airless. Along the posterior margin there was a cavity $1\frac{1}{2}$ in. \times $\frac{3}{4}$ in. with well-defined wall which contained a grey-white débris. The lower lobe was also completely consolidated with the exception of small marginal areas, and there were many small white patches and strands of grey hepatization. There were some twelve

to fifteen foci of breaking down lung tissue in the solid portion, suggestive of commencing cavity formation. Microscopically, the section showed intense packing of the lung tissue with small round cells, with complete obscuration of the alveolar arrangement in places. In addition, there was a considerable exudate of lymph and evidences of proliferation of the fixed connective tissue cells. The whole picture was one of broncho-pneumonia, which had advanced in places to wide necrosis and abscess formation (Plate 6).

There was an abscess of the lung in another case (13), which will be referred to later.

The remaining two cases with prominent respiratory manifestations were examples of broncho-pneumonia, and in one of them (2) there was an infarct with a pleural effusion.

Circulatory System.

A pulse slow in proportion to the temperature is one of the most characteristic features of paratyphoid, and the bulk of the cases have a rate lying between 70 and 90. In the severe cases, however, the pulse is rapid, and there is no single sign so significant and prophetic. If the pulse is recorded on the same chart as the temperature this feature is brought out clearly. Whereas in the ordinary case of paratyphoid the tracing of the pulse is well below that of the temperature, in more severe conditions the pulse tracing rises, first intermingling with the temperature record and then rising more and more above it. Inspection of Chart II illustrates this point well. The pulse tracing at first blended with but soon rose above the temperature tracing, and was from the first prophetic of evil, keeping at a high rate while the temperature was intermitting. The same point is well illustrated by Case 1; the patient was toxic and had broncho-pneumonia from infection by paratyphoid A. On the seventeenth day there was improvement both in pulse and temperature. On the twentieth day the temperature rose moderately, but the pulse tracing rose above it, and on the twenty-fourth day perforation occurred.

This tachycardia during the active stage of the disease only holds its significance so long as the temperature is above 100° F. It must be carefully distinguished from the tachycardia of convalescence, which has features of its own.

The pulse was soft and in six of the seventeen cases dicrotic. The apex beat in paratyphoid is not infrequently difficult to palpate, and in such cases with the polygraph a cardiogram can with difficulty be obtained. This is in keeping with the flabby and pale condition of the cardiac muscle sometimes found. Displacement outwards of the apex beat was not a marked feature, even when the heart was failing. Endocarditis was not once recorded.

The blood pressure varies. The highest recorded systolic pressure in this series was 130. It is more commonly 115-110, or even lower. The difference in reading between the diastolic and systolic pressure seemed small.

In three cases special study was made of the circulation by Captain Marris.

CHART II

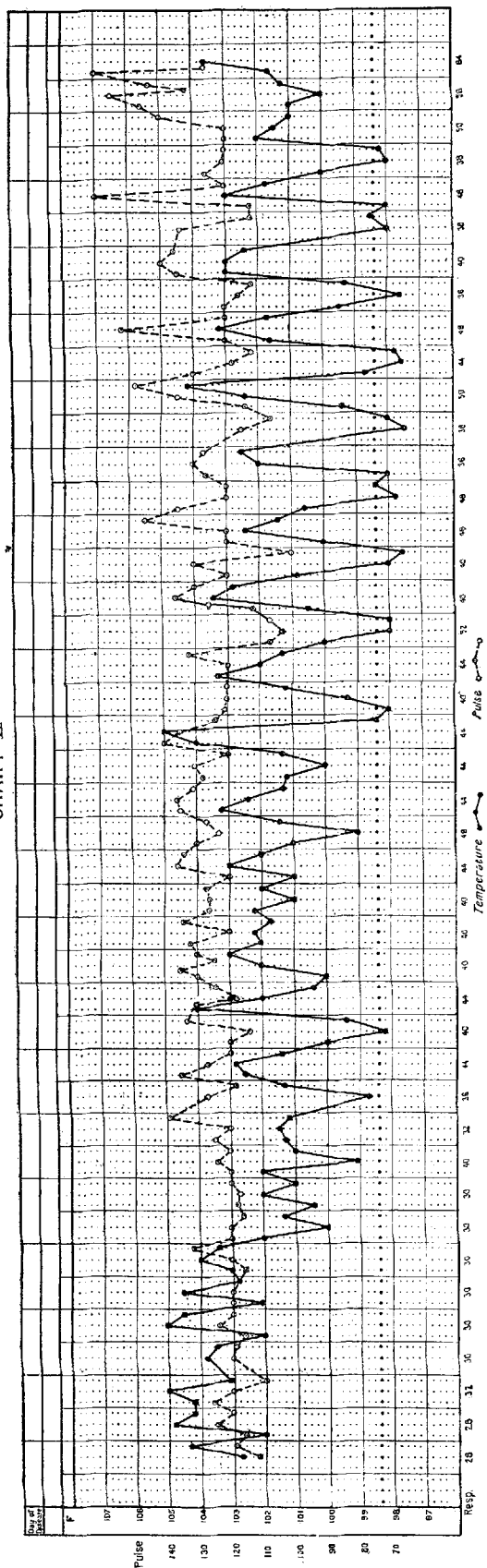
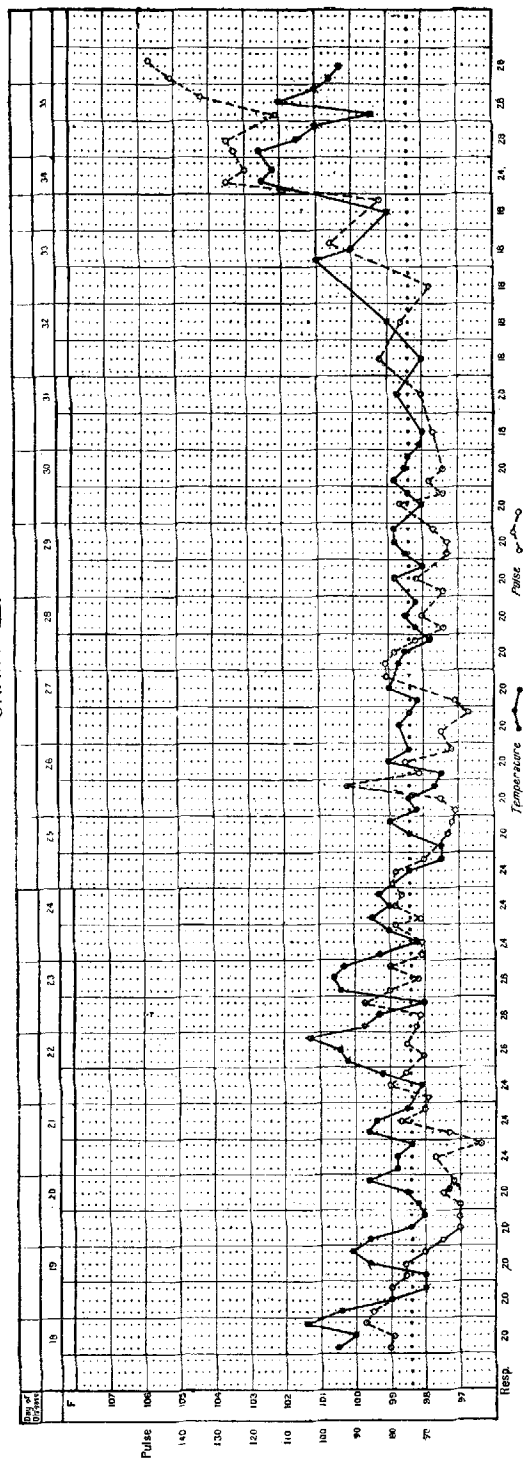


CHART III



Case 15. Pulse 90–125 dicrotic. Blood pressure 110–115 throughout. A few days before death ventricular extra-systoles occurred. This was a very toxic case which developed a suppurative parotitis, probably due to a very dry mouth and pharynx.

Case 9. Pulse 120–130 dicrotic. Blood pressure 120–130. Vessels thick and tortuous. On the thirty-fourth day showed heart-block as well as alternation of pulse. Periodic respiration during the three days preceding death. Heart, though large and fatty, did not show enlargement to percussion during life.

Case 16. Pulse mounted from average of 100 to 120 and 130, and reached 168 before death. It was dicrotic throughout. The systolic blood pressure was 95, dropping to 75 twenty-four hours before death with a diastolic of approximately 50. Heart *post mortem* was pale, flabby, and rather small.

When the condition of the patient was grave $\frac{1}{100}$ gr. strophanthin intravenously reduced the pulse from 168 to 145 in fourteen minutes, and later a second dose reduced it from 145 to 125.

The Spleen.

In eight cases the spleen was found enlarged *post mortem*; in six of these the enlargement had been detected during life, and in two it had not. In one case (No. 15) the spleen was half the normal size.

In the two following cases there were abscesses of the spleen.

Case 14, Chart III. Admitted on the eighteenth day of disease complaining of pains all over, which were worse on left side of the chest and on deep breathing; abdomen not distended; spleen not palpable, but its region was tender; the pulse was slow, fever slight, and the general aspect was one of mild infection. On the twenty-second day there was a sharp pain at the left base, increased by breathing, but no physical signs except weakness of breath sounds. The pain passed and the general condition improved until suddenly, on the thirty-fourth day, the patient vomited, the temperature rose to 102.6° and the pulse to 128. Twelve hours later there were pain and rigidity on the left side of the abdomen, and the general condition, as shown by cyanosis and weak rapid pulse, was worse. On the thirty-fifth day there was falling temperature with rising pulse, and hiccough supervened. On the thirty-sixth day death ensued.

Post mortem there were no macroscopic lesions in the intestine. The spleen (Plate 7), however, presented the following features. It was considerably enlarged and dark red in colour. The upper pole was adherent to the stomach, and the lower pole was fixed to the colon. The lower pole was converted into an abscess cavity of the size of a hen's egg and contained thick curdy pus. Half an inch above and anterior to this abscess the surface of the spleen was mottled white and purple, and beneath this area another small abscess was

found. Towards the hilum there was a third abscess cavity an inch in diameter and of irregular outline. The remaining pulp was congested and friable.

There was no perforation and no signs of peritonitis, and it is not quite clear why this patient died and so quickly.

Bac. paratyphoid B was obtained from the splenic abscess and pulp, and from the bile.

In life, cultures from blood, stools, and urine were negative. Agglutination was positive with paratyphoid B.

The full table was as follows :

	Dilutions	50	100	250	500	1,000	2,500
19th day	{ Typhosus	+	+	+	—	—	—
	{ Para. A	—	—	—	—	—	—
	{ Para. B	+	+	+	+	—	—
24th day	{ Typhosus	+	+	+	+	+	—
	{ Para. A	—	—	—	—	—	—
	{ Para. B	+	+	—	—	—	—

This case is an interesting example of paratyphoid without the usual localization in the intestine.

Case 7, Chart IV. Admitted on the ninth day ill, but only slightly toxic, with a characteristic tongue, a flat abdomen, diarrhoea and typical stools, but no spots and no splenic enlargement. Except for a persistence of the diarrhoea, progress was continuous and satisfactory until the twenty-third day, when perforation of an ulcer in the sigmoid occurred. There was a sudden onset of acute abdominal pain. Two hours later the abdomen was distended, motionless, and diffusely tender, and the liver dullness was absent. Operation five hours after the onset disclosed gas and faeces in the peritoneal cavity and three perforations in one necrotic ulcer in the sigmoid. The ulcer was sutured. Death occurred thirty-six hours later. It will be observed that perforation took place after an afebrile interval of six days. The patient had been cautiously fed.

At the autopsy there were six clean, healing ulcers of the ileum near the caecum. The large gut was thickly studded from caecum to rectum with punched-out ulcers, several of which were near perforation, besides the one which had given in the sigmoid.

The spleen contained two abscesses, each the size of a pigeon's egg. The bases of the lungs were engorged. Other organs called for no comment.

The bacteriological findings were as follows : blood culture was negative on the tenth day ; three stools were negative on the tenth, eleventh, and twelfth days ; the urine was negative on the tenth day, but positive on the sixteenth day.

Agglutinations were :

10th day	{ Typhosus	+	1 in 250
	{ Para. A	—	
	{ Para. B	+	1 in 50
14th day	{ Typhosus	+	1 in 200
	{ Para. A	—	
	{ Para. B	+	1 in 500

Post mortem both the bile and the spleen gave positive cultures of paratyphoid B.

The Intestines.

The small gut. In one case of paratyphoid A infection (No. 1) the ulceration of Peyer's patches was very marked, whilst in the other (No. 2) there were obvious signs of the healing of recent ulcers in the intestine when the patient died from a subsequent typhoid infection. In twelve out of the fourteen paratyphoid B infections the small intestine was affected to a varying extent. Usually the Peyer's patches were deeply ulcerated, and the solitary lymphoid nodules were raised and varied in size from pinheads to large peas. The lower two feet of the ileum were the constant seat of the disease, and the neighbourhood of the ileo-caecal valve was the area the most intensely affected. The floor of the ulcers was constituted by the muscular coat or the peritoneum, and the edges of the ulcers were usually undermined and in all cases thickened (Plate 8).

In two cases the small gut showed no lesions. One of them (No. 14) has been already referred to. In the other (No. 9) the large intestine was extensively ulcerated from the caecum to the pelvic colon. In a third case (No. 13), to be referred to later, there was no ulceration though Peyer's patches were raised (Plate 9).

Large intestine. Judging from this series of cases the disease falls with more force on the large intestine in paratyphoid than in typhoid. This is shown by the frequency and extent of the ulceration in the caecum, appendix, and colon. Thus in ten instances of paratyphoid B infection the large intestine from the caecum to the pelvic colon was involved, the ulceration being most marked in the caecum and the colon as far as the splenic flexure. In the milder examples the lymphoid nodules were raised, hyperaemic, and umbilicated, whereas in the severer forms there were ulcers of varying sizes and depths. A characteristic condition is shown in Plate 10, where the large bowel is riddled by small ulcers, some of which have coalesced. Notwithstanding the marked distribution of ulcers in the large intestine, it is noteworthy that there were no clinical manifestations especially suggestive of it. Moreover, in typhoid the large bowel is more often affected than is commonly supposed. The condition of the large intestine is not mentioned in two post-mortem records, and in the remaining two cases (Nos. 13 and 14) of paratyphoid B infection, in which neither the large nor the small intestine was ulcerated, there were abscesses in the liver and the spleen respectively.

Appendix. The appendix was a focus of mischief causing peritonitis in three cases. In two of these (Nos. 6 and 13), in which paratyphoid B was the cause, it was acutely inflamed. In No. 6 there was gangrene of the appendix, at the base of which there was a perforated ulcer. In the other case (No. 13)

CHART IV

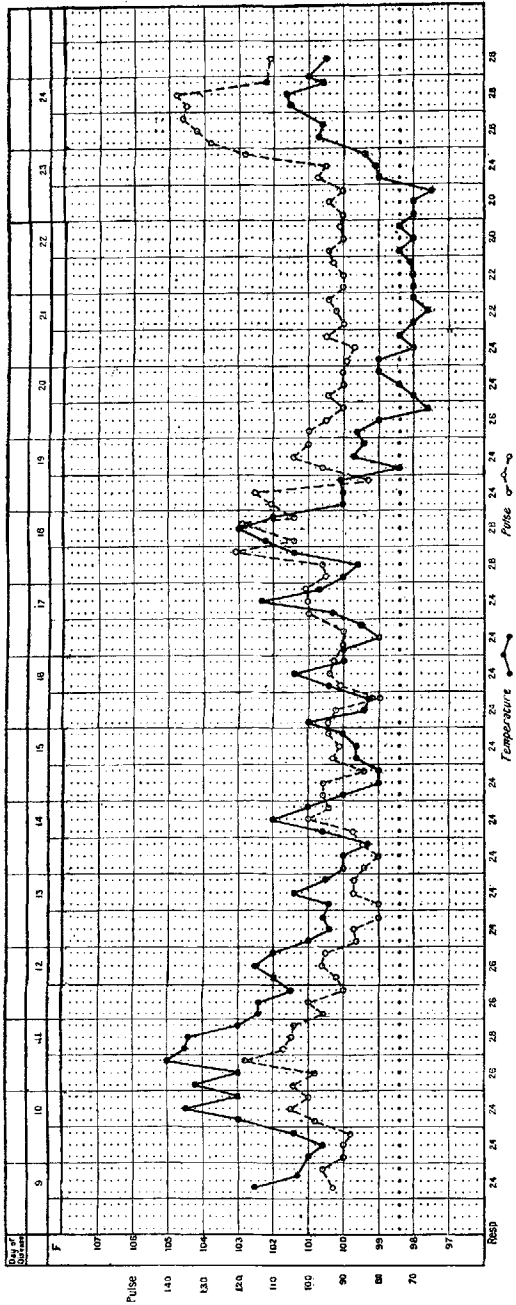
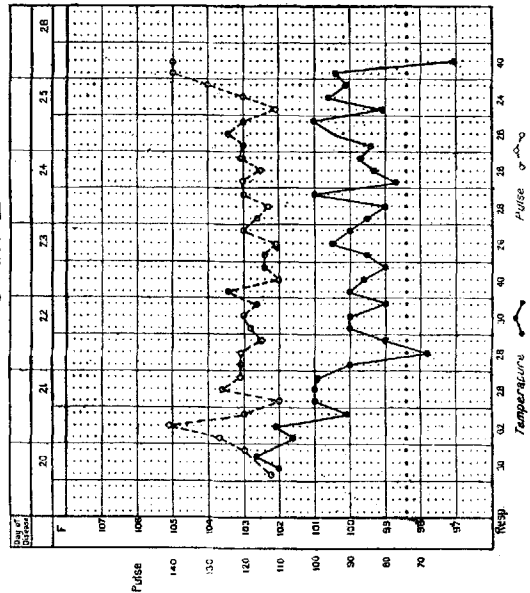


CHART V



the tip of the appendix was intensely inflamed, general peritonitis and portal pyaemia had followed, and paratyphoid B was isolated from the pus both in the right iliac fossa and the liver.

The appendix was the seat of the perforation of a typhoid ulcer in one case of paratyphoid A.

These three cases are worthy of more detailed description.

Case 6, Chart V. Admitted on twentieth day, drowsy, flushed, and toxic; abdomen slightly distended and painful; pulse rapid (110-120) and soft; heart and lungs showed no abnormal signs. On the twenty-fifth day more abdominal pain with repeated vomiting, but the abdomen moved and was not distended. The vomiting consisted of bile and, later, of coffee grounds material, and death ensued on the twenty-sixth day.

At the autopsy the peritoneal cavity contained thin, purulent fluid, and its surface was injected everywhere. The appendix was gangrenous in its proximal two-thirds and its mesentery also. There was severe and deep ulceration for five feet above the ileo-caecal valve and affecting the solitary follicles rather than Peyer's patches. Several ulcers were down to the peritoneal coat. Mesenteric glands were of the typical chocolate colour.

The blood in life and the bile after death both gave a positive culture of paratyphoid B.

Case 13, Chart VI. Admitted on the eleventh day, jaundiced and looking ill; the tongue was suggestive; the abdomen tumid and tender, especially over the gall-bladder; neither liver nor spleen was felt; there were a few spots. Pulse throughout indicated a severe infection. Bowels were constipated, and the motions contained bile. Patient became increasingly toxic, and abdominal tenderness became more marked. On the twenty-sixth day a tumour was felt in the right lower quadrant of the abdomen and at operation pus was found, apparently connected with the appendix, and was freely drained. There was only a transitory improvement after the operation. The jaundice deepened, wasting and weakness increased, and death occurred on the fifty-seventh day.

At the autopsy there was diffuse peritonitis due to a gangrenous appendix bound down in an abscess cavity in the pelvis. The liver was enlarged, and contained multiple abscesses. A liver abscess had penetrated the right diaphragm and produced an abscess in the lower lobe of the right lung and an empyema at the base of the right pleura. Peyer's patches were prominent, but there was no definite ulceration in small or large intestine.

The bacteriological findings were:

Blood culture negative on twelfth day. Stool and urine cultures negative.

Agglutinations with all three organisms were negative in all dilutions from 1 in 50 to 1 in 2,500 on two separate occasions.

Pus from the abscess showed positive culture of paratyphoid B and so did the bile.

This case is remarkable in many ways. Jaundice was a feature. The

CHART VI

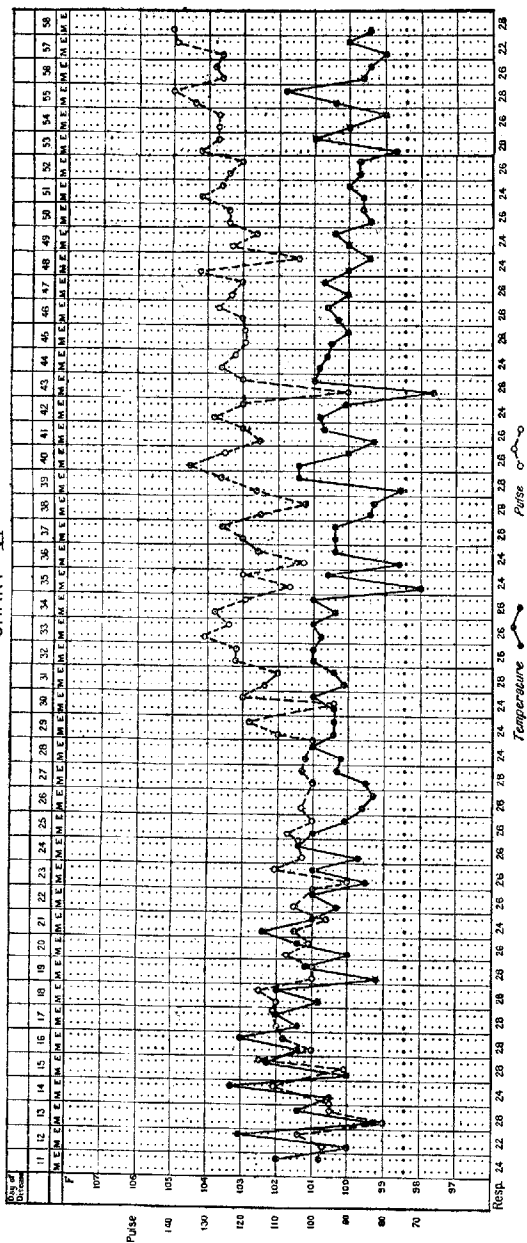
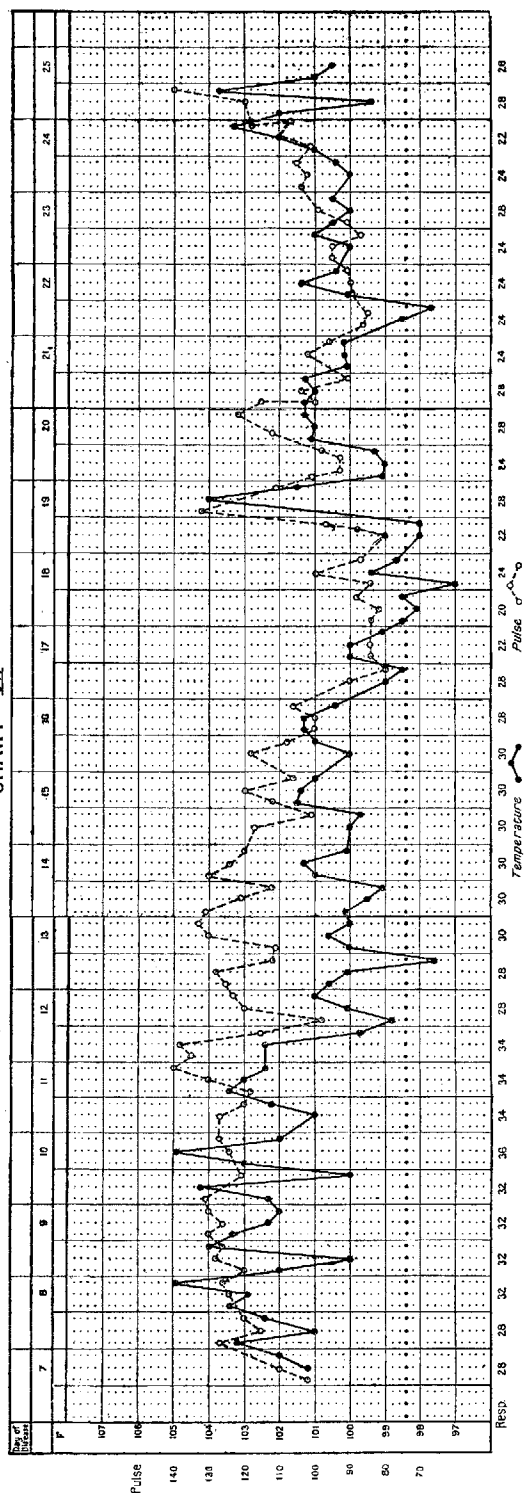


CHART VII



brunt of the disease had fallen on the appendix, and manifestations in the rest of the intestine were ill-defined. There was no acute perforation, but a secondary portal pyaemia and abscesses in the liver and lung. The blood, urine, and stool cultures and agglutinations were negative, supporting the view that negative bacteriological results are not so certain a guide as positive ones. These negative results are curious in the light of the fact that the pus from the liver and the bile gave positive cultures of paratyphoid B. An alternative explanation suggests itself, viz. that an acute appendicitis supervened at the end of a mild attack of paratyphoid fever, that the portal pyaemia was the result of the appendicitis, and that the liver abscesses were subsequently infected by paratyphoid from the biliary passages. In support of this view there was a history of two attacks of severe abdominal pain during the preceding twelve months.

Case 1, Chart VII is an example of paratyphoid A ending fatally from a perforation of an ulcer in the appendix. The onset was gradual, with malaise, fever, and diarrhoea. The patient was admitted on the seventh day, ill and toxic, pulse 110-120, tongue typical, abdomen not distended, spleen not enlarged, a few spots, and signs of bronchitis. On the ninth day spots were profuse, and broncho-pneumonia had supervened, and the general condition was worse, but there was no distension. On the sixteenth day some improvement was apparent, but on the nineteenth day there was a rigor and further infection. On the twenty-first day there was improvement in the lungs, pulse, and temperature. On the twenty-third day there were symptoms of peritonitis, but no pain or tenderness, and the clinical picture did not resemble that of an ordinary ruptured appendix. Death occurred on the twenty-fifth day. Blood culture on the seventh day showed paratyphoid A. The complete bacteriological findings are to be found in Table I.

Autopsy—general peritonitis. Some thin discoloured pus in the pelvis. The appendix pointed downwards and in its proximal third there was a perforation caused by a paratyphoid ulcer, above and below which the tissues were apparently normal (Plate 11). Peyer's patches were ulcerated above the ileo-caecal valve and in the caecum the follicles were also slightly ulcerated. The lungs showed broncho-pneumonia.

There were thus five cases of peritonitis, two caused by inflamed appendices, two by acute perforations, and in one the cause was undiscovered, though it was thought to be a perforation and was operated on for such. Of the perforations, one was situated in the appendix and the other in the sigmoid.

Haemorrhage contributed materially to the death of four patients (Nos. 8, 9, 15, and 16). In one of these (No. 8) the death occurred on the fourth day in hospital, and on the twentieth day of the disease, from severe and repeated bleeding. Two died on the thirty-eighth and thirty-ninth days of the disease respectively, and one not earlier than the thirty-fifth day. All four cases were toxic. In three of them both large and small intestines were ulcerated, and

in a fourth the ulceration was confined to the large gut. In no instance could any focal origin of bleeding be confidently determined.

There were two cases of mixed infection, viz. (a) paratyphoid B with cerebro-spinal meningitis, the organism of the former being isolated from the blood and stool and that of the latter from the cerebro-spinal fluid; (b) the other (2) was specially interesting for the following reasons:

(a) The patient had a double infection by the *Bac. paratyphosus* A and the *Bac. typhosus*.

(b) He had thrombosis of the left femoral and left external iliac veins.

(c) Four relapses occurred during the course.

(d) In the last relapse he had pulmonary infarction, and death was due to the subsequent severe lung affection on the 127th day from the onset.

A full account of the bacteriological examinations is given in Table I (Case 2). The following points are noteworthy: The patient had had no protective inoculations. Admitted on the twelfth day of the illness, the patient appeared to be typical of a rather severe enteric group infection, and his blood gave a pure culture of *Bac. paratyphosus* A. The serum on this day and on the eighteenth day strongly agglutinated the stock paratyphoid A bacillus, and gave no reaction with *Bac. typhosus* or *Bac. paratyphosus* B. By the twenty-second day the patient was obviously improving, and during this time he had had a swinging temperature (rather characteristic of paratyphoid A infection) from 99° to 102°. On the twenty-third day, however, the temperature range became steadier, remaining between 102° and 104° for five days. On the twenty-fourth day the serum agglutinated *Bac. typhosus* as well as *Bac. paratyphosus* A. It gave the same reaction on the twenty-ninth day, but the reaction with *Bac. paratyphosus* A had much diminished. On the twenty-seventh day thrombosis of the left femoral vein was first noted. The duration of this primary attack of fever lasted forty-eight days.

The patient had four relapses with four, twenty, sixteen, and ten days' pyrexia respectively. During the second relapse the patient was given two injections of paratyphoid A vaccine without obvious effect. In the middle of the third relapse a blood culture was negative. At the post-mortem a pure culture of *Bac. typhosus* was grown from every viscus examined (gall-bladder, spleen, mesenteric gland, and thrombosed vein), thus proving the presence of a second infection.

The date of the second bacillary invasion is not quite clear. The agglutination reactions suggest that it was before the twenty-fourth day, but not much before the eighteenth day; also the temperature range altered on the twenty-third day.

Thus it would seem likely that when the patient came into hospital he had reached the twelfth day of a paratyphoid A attack and was in the midst of the incubation period of typhoid; that for a while the two infections reigned together, and later the paratyphoid A disappeared, leaving the typhoid to reign alone. The relapses were thus probably due to *Bac. typhosus*.

The causes of death may be summarized as follows :

Perforation	2 cases
Peritonitis from infected appendix	2 „
Haemorrhage	2 „
Haemorrhage and toxaemia	3 „
Toxaemia	4 „
Pneumonia	2 „
Splenic abscess	1 „

These represent a death-rate of rather over 4 per cent. for paratyphoid B and under 1 per cent. for paratyphoid A.

Complications.

Apart from perforation, haemorrhage, and pulmonary manifestations, these cases seem to point to a tendency to pus formation. Thus abscesses of the spleen were found twice (Nos. 7 and 14); round appendix twice (Nos. 6 and 13); in the liver once (No. 13), and of lung twice (Nos. 3 and 13); and empyema occurred in connexion with one of the lung abscesses.

Beyond these there was one instance (No. 2) of left femoral thrombosis with pulmonary infarct and left pleural effusion.

No small part of the value of these records lies in the complete bacteriological observations, for which we are greatly indebted to Captain J. L. Wood and Captain W. H. Tytler.

Treatment.

Followed the usual lines. Two patients were treated with paratyphoid sensitized vaccine, one (No. 2) as above, and the other (No. 3) received 250 millions on the eighteenth, 375 millions on the nineteenth, and 500 millions on the twentieth day. Neither benefit nor the reverse apparently followed.

Laparotomy was performed on four of these cases, on two for peritonitis due to suppuration round the appendix (Nos. 6 and 13), and on two for perforation. In one of the latter (No. 10) the evidence at the onset was vague, and operation was not undertaken until twenty-four hours later, and even then the ulcer which caused the peritonitis was not found; but in the other (No. 7) the abdomen was opened within five hours of the commencement of symptoms. The results in these two cases are in keeping with those for perforation generally at the hospital in which these cases occurred. So far there has been no recovery, and this notwithstanding that the external conditions give every chance for success in that the patients are constantly under the eyes of skilled observers and the surgery is in the hands of a skilled abdominal operator, Major Davies-Colley.

It is the internal conditions which are so unfavourable. For one ulcer which is through there may be several which are nearly so, and the reactive power, both local and general, is down to vanishing point. This was so, for

instance, in Case 7, in which there were several ulcers almost as bad as the one which had actually perforated. It would seem that the only cases in which we can expect success are those in which some odd ulcer has gone deeper than its fellows and perforated the weak link in a chain which otherwise has some strength left; and it is these fortunate cases which fully justify resort to operation when perforation has been diagnosed, and provided sufficiently skilled surgical aid is available.

TABLE I. *Summary of Symptoms and Signs and*

No.	Protective Inocula- tions against Typhoid.	Onset.	Condition on Admission.	Tongue.	Pulse.	Rash.	Abdomen.	Spleen.
1.	+2	Gradual, with diarrhoea and great weakness. No headache. No epistaxis	8th day. Toxic, pinched, and cyanosed. T = 101°. P = 108. R = 28.	Furred and sticky	110-120. Not dicrotic.	8th day. A few spots on abdomen. 14th day. Profuse	Not distended or tender on admission	Never enlarged
2.	0	Headache, cough, abdominal pain, diarrhoea. No epistaxis	13th day. Drowsy and mentally weak. T = 102.8°. P = 108. R = 28.	Dry and furred	Small and compressible. Not dicrotic	13th day. A few spots on back. 20th day. A fresh crop.	Not distended or tender on admission	14th day. + (felt). And again during first relapse
3.	+2	History not obtained	8th day. Lethargic and toxic. Typical appearance of severe case. T = 103°. P = 110. R = 28.	Dry, brown fur. Shiny tip and edges	Big volume. Soft and dicrotic	No spots seen	Not distended. No tenderness	8th day. + (easily felt)
4.	+2	History could not be obtained	4th day. Seemed a mild case, but mentally peculiar, refusing to speak. T = 100°. P = 84. R = 24.	Dry, thickly furred. Edges clean	Big volume. Not dicrotic	Big, markedly raised spots (? Para. B)	Rather distended. Moved well and was not tender	4th day. + (just felt)
5.	+2	General weakness. Chilly. Abdominal pain. No epistaxis or headache	18th day. Appeared severe. Vomiting, abdominal pain and diarrhoea. T = 103°. P = 120. R = 32.	Thick fur	Low tension	21st day. Spots present	Soft, but distended and tender	+ (not felt)

Clinical Course, with the Bacteriological Reports.

<i>Lungs.</i>	<i>Stools.</i>	<i>Course.</i>	<i>Bacteriological Reports.</i>
General bronchitis. Later severe	Typical loose brown. Some diarrhoea	12th day. Broncho-pneumonia 19th day. Rigor 23rd day. Peritoneal appearance. Abdomen quite flaccid 25th day. Died	<i>During life.</i> Blood culture, 7th day = Bac. Para. A Agglutination reactions not done <i>Post mortem</i> , 25th day. Culture from gall-bladder = Bac. Para. A
13th day. Bronchitis 114th day. Gradual increase in signs in left lung, with blood - spitting and acute pain in left side of chest	13th-27th days. Had slight diarrhoea. After this had constipation	23rd day. ? Fresh infection 28th day. Left femoral thrombosis 40th day. General condition good 53rd day. Relapse 87th day. Relapse 114th day. ? Infarct of left lung and relapse 121st day. Right heart dilated 128th day. Died	<i>During life.</i> Blood culture, 12th day = Bac. Para. A. Blood culture, 97th day (10th day of relapse) = neg. Agglutinations:— ¹ 12th day, T = 0, A = 1/500, B = 0 18th day, T = 0, A = 1/500, B = 0 24th day, T = 1/250, A = 1/500, B = 0 29th day, T = 1/250, A = 1/50, B = 0 36th day, T = <1/50, A = <1/50, B = <1/50 <i>Post mortem</i> , 126th day. Cultures from gall-bladder, spleen, mesenteric gland, and from a thrombus in the left exterior iliac vein grew Bac. typhosus
8th day. Nil 15th day. Bronchitis 21st day. Broncho-pneumonia 23rd day. Dullness + overright base; ? empyema. Chest explored 27th day. Dullness at both bases	Loose, at times undigested. 1-2 per diem until two days before death, when severe diarrhoea started	Gradual involvement of both lungs 23rd day. Swinging temperature (97°-103°) from now onwards: ? rapidly advancing tubercular affection. Appearance not typhoidal. Patient was at times fairly bright. 3 doses of Para. B vaccine without appreciable effect 29th day. Died	<i>During life.</i> Blood culture, 8th day = Bac. Para. B. 23rd day, fluid from lung puncture sterile. No tubercle bacilli in sputum Agglutinations:— 8th day, T = 1/100, A = 0, B = 0 15th day, T = 1/250, A = 1/25, B = 1/500 Smear made from material obtained by lung puncture showed no bacilli <i>Post mortem</i> , 29th day. Cultures from gall-bladder and from lung grew Bac. Para. B
4th day. Nil. R = 24 6th day. R = 34 8th day. Crepitations and tubular breathing at right base and less so at left base	Loose brown. Not frequent	6th day. Definite turn for the worse, and case took on the aspect of noisy delirious lobar pneumonia 11th day. Died	<i>During life.</i> Blood culture, 7th day = Bac. Para. B Agglutinations:— 7th day, T = 1/250, A = 0, B = 1/100 11th day, T = 1/500, A = 0, B = 1/25 The patient's serum agglutinated strongly the bacillus isolated from his blood <i>Post mortem</i> , 11th day. Culture from gall-bladder grew Bac. Para. B
18th day. Nil 21st day. Bronchitis	Diarrhoea throughout. 9-11 per diem	Increasing toxæmia 21st day. Died	<i>During life.</i> Blood culture, ? 16th day = Bac. Para. B. Stool culture, ? 16th day = Bac. Para. B Agglutinations:— T = 1/3,000, A = <1/100, B = 1/4,000 <i>Post mortem</i> , 21st day. Culture from gall-bladder grew Bac. Para. B. Culture from lung showed no organisms of enteric group

¹ These cases, with the exception of No. 16, were in this hospital before standard emulsions had come into general use. The various dilutions mentioned therefore cannot be compared. In most cases, however, the same emulsions were used for all the tests made on a particular case.

TABLE I

No.	Protective Inocula- tion against Typhoid.	Onset.	Condition on Admission.	Tongue.	Pulse.	Rash.	Abdomen.	Spleen.
6.	+ 2	Diarrhoea, vomiting, abdominal pain, epistaxis	? 20th day. Typical of a severe case of enteric group infection. T = 102°. P = 112. R = 28.	Furred	112-120. Low tension	A few spots on abdomen	Tender and slightly dis-tended. 25th day. Not dis-tended	+ (felt)
7.	+ 1	Headache, back-ache, pain in abdomen and limbs. Shivering, diarrhoea. No vomiting. No epistaxis	9th day. Toxic. T = 103°. P = 96. R = 24.	Dry, furred	Dicrotic. Very soft	No spots seen	9th day. Nil. 11th day. Elastic and slightly dis-tended	+ (not felt)
8.	+ 2	History could not be obtained	? 20th day. Toxic, delirious. Typical of a severe typhoid case. T = 101.8°. P = 112. R = 24.	Dry, brown. Shiny at tip. Typical	Dicrotic. Very soft	A few big spots	Fullness. No ten-derness	+ (not felt, but markedly enlarged to per-cussion)
9.	+ 2	Gradual. Ab-dominal pain. Three weeks diarrhoea. Epi-staxis on two occasions	19th day. Head-ache, abdo-minal pain and diarrhoea. Typical group infection, prob-ably Para. B. T = 103.8°. P = 76. R = 20.	Thick fur	Dicrotic.	Several rosespots of large para-typhoid type. 30th day. Spots very notice-able	Tumid. No ten-derness	+ (not felt). 28th day. (felt)
10.	+ 2	General weak-ness, slight diarrhoea and abdominal pain. Severe headache. No epistaxis	9th day. Dusky with malar flush. Head-ache, mentally clear. Moder-ately severe and typical paratyphoid. T = 100.6°. P = 84. R = 20.	Dry, cracked, clean at edges and tip	Dicrotic. Good volume. Very soft	A few big, well-raised spots (? Para. B)	Not dis-tended. Nil ab-normal	9th day. Not en-larged. 12th day. + (felt)

(continued).

<i>Lungs.</i>	<i>Stools.</i>	<i>Course.</i>	<i>Bacteriological Reports.</i>
?20th day. Nil. Never had abnormal signs	Diarrhoea throughout. 2-6 per diem	24th day. Toxic, but general condition seemed improved 25th day. Vomiting, abdominal pain, abdomen moved well and liver dullness normal 26th day. Died	<i>During life.</i> Blood culture, ?20th day = Bac. Para. B Agglutination reaction of serum not tested <i>Post mortem</i> , ?26th day. Culture from gall-bladder grew Bac. Para. B
9th day. Bronchitis. This cleared up later	Typical. Diarrhoea after 11th day. 3-5 per diem	9th to 22nd days. Steady improvement to convalescence 20th day. Pea-soup stools 23rd day. Sudden abdominal pain, peritonitis, laparotomy 25th day. Died	<i>During life.</i> Blood culture, 10th day = neg. 3 stool cultures and one urine culture from 10th to 13th days = all neg. Urine culture, 15th day = Bac. Para. B Agglutinations:— 10th day, T = 1/250, A = 0, B = 1/50 15th day, T = 1/250, A = 0, B = 1/500 <i>Post mortem</i> , 25th day. Cultures from gall-bladder and spleen grew Bac. Para. B Culture from lung showed no enteric group bacilli Smear from lung showed no Gram-negative bacilli
Nil	Typical greenish-brown. 1-2 per diem	?22nd day. Slough (?) passed ?23rd day. Haemorrhages, large and repeated ?24th day. Died suddenly	<i>During life.</i> Blood culture, ?20th day = Bac. Para. B. Cultures from stool and urine of same day also gave Bac. Para. B Agglutinations:— ?20th day, T = 1/320, A = 0, B = 1/540 <i>Post mortem</i> , ?24th day. Culture from gall-bladder grew Bac. Para. B
19th day. Clear. No bronchitis throughout	Constipated, until 3 days before death	22nd to 33rd days. Increasing toxæmia 33rd day. Tender left side of abdomen, but no rigidity 34th to 36th days. Haemorrhages 38th day. Heart failing 39th day. Died	<i>During life.</i> Blood culture, 20th day = neg. Stool and urine cultures on 20th day also negative Agglutinations:— 20th day, T = 1/1,000, A = 0, B = 1/1,000 26th day, T = 1/20,000, A = 0, B = 1/20,000 35th day, T = 1/1,000, A = 0, B = 1/1,000 <i>Post mortem</i> , 39th day. Culture from gall-bladder grew Bac. Para. B Cultures from spleen and lung were negative to enteric group
No involvement of lungs	1-2 per diem	12th day. Drowsy. Abdomen hollowed 13th day. Had had two doses of Para. B vaccine without obvious effect 14th day. Toxic. Abdominal pain 15th day. Pain and rigidity over lower abdomen, especially left iliac fossa. Vomited. ? Shut off perforation. ? Peritonitis without perforation 16th day. Laparotomy. No perforation found. General peritonitis 17th day. Died	<i>During life.</i> Blood culture, 9th day = Bac. Para. B. Stool culture, 11th day = Bac. Para. B Agglutinations:— 9th day, T 1/100, B = 1/100 <i>Post mortem</i> , 17th day. Culture from gall-bladder grew Bac. Para. B. Cultures from the spleen and from a sub-peritoneal haemorrhage grew Bac. coli. A culture from a haemorrhagic area in lung grew staphylococci, but a smear from the lung showed Gram-negative bacilli

TABLE I

No.	Protective Inocula- tions against Typhoid.	Onset.	Condition on Admission.	Tongue.	Pulse.	Rash.	Abdomen.	Spleen.
11.	+1	History could not be obtained	10th day. Flushed, delirious, suggestive appearance. T = 103°. P = 124. R = 28.	Dry, furred, cracked, typical	Soft, but not dicrotic	Nil definite (had scabies)	Soft, tumid, tender	Not made out to be enlarged
12.	?	History could not be obtained	5th day. Flushed, drowsy, lethargic. Very toxic. T = 103°. P = 100. R = 32.	Dry, brown, very typical	Full, but easily compressible	No spots seen	Distended. Tender in region of spleen	+ (not felt)
13.	0	Anorexia, constipation, abdominal pain. No diarrhoea, vomiting, or epistaxis. (Two attacks of severe abdominal pain in last 12 months)	11th day. Slightly jaundiced, looked ill. T = 102°. P = 98. R = 24.	Fairly moist. Furred, but clean at edges	Full volume, medium tension	A few suspicious spots	Tumid and tender over gall-bladder. 13th day. Markedly distended	Not enlarged at first. 23rd day. + to percussion
14.	+1	Headache, pains in limbs, joints, and abdomen. Vomited. No diarrhoea	18th day. Quite clear mentally. Pains all over, especially in left side of chest on drawing a deep breath. T = 100°. P = 80. R = 20.	Rather dry, but quite clean. Not typical	Low tension	No spots seen	Not distended. Especially tender over splenic area	Not made out enlarged

(continued).

<i>Lungs.</i>	<i>Stools.</i>	<i>Course.</i>	<i>Bacteriological Reports.</i>
Slight bronchitis at first. Later severely involved	Constipated throughout	12th day. Toxic. Lungs seemed choked at bases, but good percussion note 14th day. ? Aspiration pneumonia at both bases. Death	<i>During life.</i> Blood culture, 10th day = Bac. Para. B Agglutinations:— 10th day, T = 1/640, B = 1/120 <i>Post mortem</i> , 14th day. Cultures from gall-bladder, a mesenteric gland, and from the blood in one of big veins near heart all grew Bac. Para. B. A culture made from pus in lung showed no bacilli of the enteric group
5th day. Occasional râles 12th day. Much bronchitis	Fluid and typical. 3-8 per diem	7th day. More toxic 15th day. Haemorrhage (less than a pint) 16th day. Very severe. Nil in lungs to explain condition 18th day. Death	<i>During life.</i> Blood culture, 6th day = Bac. Para. B Agglutinations:— ² 8th day, T = 1/120, B = 1/250 11th day, T = 1/25,000, B = 1/12,500 14th day, T = 1/250,000, B = 1/50,000 17th day, T = 1/125,000, B = 1/250,000 <i>Post mortem</i> , 18th day. Cultures from spleen and gall-bladder both grew Bac. Para. B. Culture from faeces grew ? Proteus
Little, if any, involvement of lungs	Alternate constipation and diarrhoea. Chiefly constipation	11th to 25th days. Constant abdominal pain, increasing distension and toxæmia 26th day. Mass felt in right iliac fossa. Abdomen soft and pulse better. Jaundice increasing. Laparotomy: shut-off abscess in appendix region opened and tubes inserted 34th day. Wound opened up and tubes reinserted 53rd to 58th days. Jaundice deepened and slowly went downhill 58th day. Death	<i>During life.</i> Blood culture, 12th day = neg. Three stool cultures and one urine culture during the 12th to 24th days were all negative Agglutinations:— 12th day, T = <1/50, A = <1/50, B = <1/50 24th day, T = <1/50, A = <1/50, B = <1/50 <i>Post mortem</i> , 58th day. Cultures from gall-bladder and from abscess in liver both grew Bac. Para. B. Culture from spleen showed no enteric group bacilli
No involvement of lungs	Decidedly constipated throughout	22nd day. Sharp pain in left lower chest 33rd day. Doing well. Uninterrupted progress 34th day. Severe vomiting and rise of temperature. Hollowed and flaccid abdomen. Later, severe pain in left upper abdomen with rigidity 35th day. Abdomen very retracted. Very weak. Hiccough 36th day. Died	<i>During life.</i> Blood culture, 16th day = neg. Agglutinations:— 16th day, T = 1/250, B = 1/500 24th day, T = 1/1,000, B = 1/100 <i>Post mortem</i> , 36th day. Cultures from gall-bladder, from spleen pulp, and from splenic abscess all grew Bac. Para. B. A culture made from blood within the heart was sterile. Serum from blood taken from heart agglutinated T = 1/500 and B = 1/500

² Case No. 12. Agglutination tests made by Dr. Inman with very dilute emulsions and heated at 55° C. This series is comparative. No information could be obtained about protective inoculation in this case. The agglutination curves suggest that he had been inoculated.

(continued).

<i>Lungs.</i>	<i>Stools.</i>	<i>Course.</i>	<i>Bacteriological Reports.</i>
No involvement of lungs	Diarrhoea throughout. 4-7 per diem	Irregular pulse, and very lethargic. End of 3rd week (?). Parotitis incised—no pus. Later increasing toxæmia and hæmorrhages. Difficulty in speaking and swallowing throughout. Died ?early in 5th week	<i>During life.</i> Blood culture, ?2nd week = Bac. Para. B. Stool culture, ?3rd week = Bac. Para. B. Three urine cultures and two stool cultures before this all negative Agglutinations:— 2nd week, T=1/250, B=<1/50 End of 3rd week, T=1/10,000, B=1/5,000 Culture from parotid swelling showed no enteric group bacilli <i>Post mortem</i> , ?early in 5th week. Cultures from gall-bladder and spleen both grew Bac. Para. B
Bronchitis	Typical. Some diarrhoea	Advancing toxæmia, with very weak and irregular pulse (110-130) 32nd day. Severe hæmorrhages 38th day. Died	<i>During life.</i> Blood culture, 17th day = Bac. Para. B. Stool culture on same day also grew Bac. Para. B Agglutinations:— 17th day, T = 1/80 (20 units), A and B < 1/50 23rd day, T=1/900 (230 units), B=1/5,500 (2,750 units) 30th day, T=1/3,500 (895 units), B = 1/24,000 (12,000 units) <i>Post mortem</i> , 38th day. Cultures from gall-bladder and spleen both grew Bac. Para. B
Basal bronchitis	Typical. Diarrhoea throughout. 3-7 per diem	Gradually increasing distension and cardiac weakness 15th day. Severe hæmorrhages 16th day. Death	<i>During life.</i> Blood cultures made on 10th and 12th days both grew Bac. Para. B. No agglutination tests were made <i>Post mortem</i> , 16th day. Culture from spleen grew Bac. Para. B
		The cerebral symptoms became prominent soon after admission and were the dominant feature throughout. Patient when admitted did not appear a severe case of paratyphoid fever	<i>During life.</i> A blood culture and a stool culture made in second (?) week of illness both grew Bac. Para. B. The cerebro-spinal fluid showed microscopically the Meningococcus intra-cellularis, which organism was also obtained on culture

TABLE

No.	<i>Peritoneum and Small Intestine.</i>	<i>Large Intestine and Appendix.</i>	<i>Mesenteric Glands.</i>	<i>The Spleen.</i>
1.	<i>Peritoneum.</i> General peritonitis. Peyer's patches ulcerated in last 18" of ileum	<i>Caecum.</i> Solitary follicles deeply ulcerated <i>Appendix</i> 2½" long: ulcer ¾" x ¼" had perforated through ulcer at its middle	Not mentioned	Not mentioned
2.	<i>Peritoneum.</i> Clean. Lower 4 feet, healed ulcers and some not quite healed	Not mentioned	Enlarged and pale	Large and pulpy. Two normal
3.	<i>Peritoneum.</i> Clean. Lower 6 feet of intestine ulcerated to submucosa and peritoneum	Not mentioned	Not mentioned	Not mentioned
4.	<i>Peritoneum.</i> Clean. Peyer's patches of ileum, marked ulceration and solitary follicles. Raised and central ulceration	Not mentioned	Not mentioned	Not mentioned
5.	<i>Peritoneum.</i> Clean. Superficial ulceration of Peyer's patches	Acute diffuse hyperaemia, prominent hyperaemic nodules. Ulceration of centre of lymphoid nodules	Not mentioned	Firm, not enlarged
6.	<i>Peritoneum.</i> General peritonitis in ileum, packed ulcers of Peyer's patches:—floor of ulcers—peritoneum	<i>Caecum.</i> Many ulcers and small ulcer at base of appendix <i>Appendix</i> gangrenous throughout length	Enlarged and chocolate coloured	1½ normal, friable
7.	<i>Peritoneum.</i> General peritonitis. Six small healing ulcers in last foot	Intense hyperaemia and ulceration from caecum to pelvic colon. Perforation of ulcer at sigmoid flexure	Enlarged and some contained pus	Abscess at each pole
8.	<i>Peritoneum.</i> Clean. In ileum Peyer's patches swollen. Solitary follicles prominent and ulcerated	<i>Caecum.</i> Many small rounded ulcers. Solitary follicles of whole large intestine ulcerated	Congested	1½ normal, firm
9.	<i>Peritoneum.</i> Recent peritonitis in left flank. No evidence of ulceration of small intestine	Large and small, clean, but ulcers from caecum to pelvic colon	Slightly enlarged. Not typical	2½ normal, friable
10.	<i>Peritoneum.</i> General peritonitis. Semi-gangrenous ileum and submucous haemorrhages	<i>Caecum.</i> Early ulceration	Not mentioned	Not mentioned
11.	<i>Peritoneum.</i> Clean. Lower 2½ feet of ileum, marked ulceration of Peyer's patches, especially at ileocaecal valve	<i>Caecum</i> to splenic flexure profusely peppered with small punched out ulcers	Enlarged, hard and typical	1¼ normal, 'red septic spleen'

II.

<i>The Liver.</i>	<i>The Kidneys.</i>	<i>The Heart.</i>	<i>Lungs and Pleura.</i>	<i>Summary.</i>
Not mentioned	Cloudy swelling	Normal	Congested and broncho - pneumonia in places	Paratyphoid A ulceration of small intestine and appendix. Perforation of paratyphoid ulcer of appendix. General peritonitis
Mottled and pale	Nil	Flabby and dilated	Left pleural effusion and consolidation of left lower lobe	1st attack—paratyphoid A 2nd attack—typhoid
Not mentioned	Cloudy swelling	Nil	Acute gangrenous pneumonia (see text)	Paratyphoid B pneumonia
Not mentioned	Nil	Nil	Right lower lobe, red-grey pneumonic consolidation	Paratyphoid B ulceration of small intestine. Acute pneumonia
Congested	Cloudy swelling	Nil	Lungs congested	Paratyphoid ulceration of small and large intestine. Heart failure
Congested	Cloudy swelling	Nil	Lungs congested	Paratyphoid ulceration of small intestine. Ulcer at base of appendix gangrenous. Appendicitis
Normal. Pus in gall-bladder	Cloudy swelling	Toxic	Hypostatic congestion	Paratyphoid B ulceration of large intestine. Perforation. Pyaemia. Laparotomy
Normal	Normal	Normal	Normal	Paratyphoid ulceration of small and large intestine. Haemorrhages
Large and fatty. Bile turbid	Fatty	Fatty	Bronchiolitis	Paratyphoid B ulceration of large intestine. Haemorrhages
Not mentioned	Cloudy swelling	Normal	Broncho-pneumonia	Paratyphoid B ulceration of ileum and caecum. ? Perforation. General peritonitis
Liver and gall-bladder normal	Normal	Normal	Early broncho-pneumonia	Paratyphoid B ulceration of small and large intestine

TABLE II

No.	<i>Peritoneum and Small Intestine.</i>	<i>Large Intestine and Appendix.</i>	<i>Mesenteric Glands.</i>	<i>The Spleen.</i>
12.	Whole of ileum packed with large ulcers: sloughs recently separated	<i>Caecum</i> to rectum mucous membrane thickened	Swollen, soft and haemorrhagic	Firm, slightly enlarged
13.	<i>Peritoneum.</i> General peritonitis. Prominent Peyer's patches. No ulceration	<i>Appendix</i> long and the last $\frac{1}{2}$ " was gangrenous	Not mentioned	$2\frac{1}{2}$ normal, not friable
14.	<i>Peritoneum.</i> Clean. No obvious macroscopic lesion in small intestine. Microscope showed 'marked hyperaemia'	Nil	Hyperaemic	Large abscess in lower pole, two other small abscesses (see text)
15.	<i>Peritoneum.</i> Clean. Last 15" of ileum, ulceration of Peyer's patches, severe at ileo-caecal valve	<i>Caecum.</i> Markedly ulcerated and extended to splenic flexure in lesser degree	Typical	Half the normal size
16.	<i>Peritoneum.</i> Clean. Diffuse superficial ulceration in lower 12" of ileum. No relation to Peyer's patches	<i>Caecum</i> to rectum, small ulcers of solitary follicles and numerous large flat ulcers, whose floor was peritoneum	Not mentioned	Normal
17.	<i>Peritoneum.</i> Clean. Very extensive and severe ulceration			

(continued).

<i>The Liver.</i>	<i>The Kidneys.</i>	<i>The Heart.</i>	<i>Lungs and Pleura.</i>	<i>Summary.</i>
Gall-bladder distended with turbid bile	Cloudy swelling	Normal	Small patch of broncho - pneumonia at base of right lower lobe	Paratyphoid B ulceration of small and large intestine
Liver enlarged and riddled with small abscesses. Gall - bladder thick, wall not enlarged. Bile clear	Not mentioned	Not mentioned	Liver abscess had penetrated right diaphragm and caused empyema and abscess in the right lower lobe of the lung	Paratyphoid B hyperaemia of small intestine. Gangrenous appendicitis. General peritonitis. Portal pyaemia
Liver normal. Gall-bladder was distended	Soft and pale	Pale and flabby	Congested	Paratyphoid B infection of small intestine. Pyaemia. Abscesses of spleen
Not mentioned	Pale	Nil	Not mentioned. Paralysis of vocal cords	Paratyphoid B ulceration of small and large intestine. Haemorrhages
Large and fatty. Gall-bladder was distended	Nil	Small and flabby	Congested. Adhesions both sides of chest, dense and universal in right side	Paratyphoid B ulceration of small and large intestine. Repeated haemorrhages

DESCRIPTION OF PLATES.

PLATE 6. Section of left lung. The anterior portion of the upper lobe is congested, though aerated; the posterior portion is consolidated, and there are two abscess cavities in the centre of areas of consolidation.

The whole of the lower lobe is occupied by broncho-pneumonic areas of consolidation, which in many places have broken down into abscess cavities. At the base of the lung a small abscess appears through the pleura.

PLATE 7. The spleen shows a large abscess cavity at its lower pole, and along the anterior margin a small cavity has been opened. Between these two cavities the spleen is mottled yellow and blue-grey, and indicates an underlying third abscess. The wall of the lower abscess is adherent to the colon.

PLATE 8. A lower portion of the ileum shows numerous ulcers, from which the sloughs have completely separated.

PLATE 9. A Peyer's patch in the ileum, showing hyperaemia and early necrosis.

PLATE 10. The ileum and caecum. The ulcers in the ileum are deep, and the sloughs have in part separated. The caecum is riddled by small ulcers, some of which have coalesced.

PLATE 11. There is an ulcer of the appendix which has a large perforation; the appendicular tissue around the ulcer was apparently healthy.



Section of Left Lung. The anterior portion of the upper lobe is congested though aerated; the posterior portion is consolidated, and there are two abscess cavities in the centre of areas of consolidation.

The whole of the lower lobe is occupied by Broncho-pneumonic areas of consolidation, which in many places have broken down into abscess cavities. At the base of the lung a small abscess appears through the Pleura.



The Spleen shows a large abscess cavity at its lower pole, and along the anterior margin a small cavity has been opened. Between these two cavities the Spleen is mottled yellow and blue-grey, and indicates an underlying third abscess. The wall of the lower abscess is adherent to the colon.



A lower portion of the Ileum shows numerous ulcers from which the sloughs have completely separated.



A Peyer's patch in the Ileum, showing hyperæmia and early necrosis.



The Ileum and Cæcum. The ulcers in the Ileum are deep, and the sloughs have in part separated. The Cæcum is riddled by small ulcers, some of which have coalesced.



There is an ulcer of the Appendix which has a large perforation; the appendicular tissue around the ulcer was apparently healthy.