

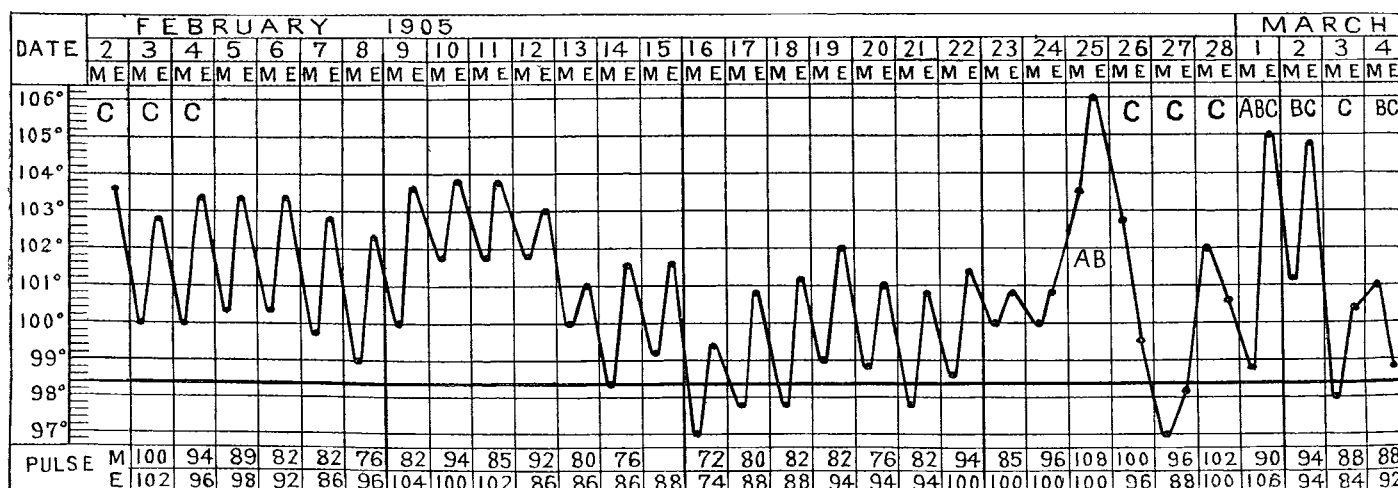
to the surface surrounding and overlying the growth. The battery supplying the current could be worn and carried about by the patient. In this manner the electric current would reach and traverse the growth from numerous points in much the same way as normal nervous impulses reach the tissues through the numberless endings of the delicate nerve fibrils lying distributed between and amongst their constituent cells.

PARATYPHOID FEVER.

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THE patient was a subaltern officer, aged 22 years, of strong and sound constitution. He had never had malaria or enteric fever. He had never been inoculated with anti-typhoid serum. On Jan. 3rd, 1905, he left Lucknow on completion of a riding course and returned to his station in Central India, taking his meals at various refreshment rooms *en route*. He first felt unwell on Jan. 26th after a long day's shooting. He complained of frontal headache and was "off his feed." He was sent out on survey duty in the district and had to stand about in water and camped out at night. During this time he felt seedy and the headache and anorexia increased and he had fever on the 30th. On the next day I went out to see him and found him in a state of high fever without rigors, but accompanied by vomiting, complete anorexia, and headache. Quinine and diaphoretics and phenacetin were given, the first at the rate of 40 grains per diem. The symptoms during the first week of the disease—i.e., from Feb. 1st to 8th—were as follows. The temperature ranged between 100° and 103·5° F., being always lowest at midday and highest in the evening (see chart). There were no rigors

pulse was very soft. The tongue remained foul and the fur somewhat browner. The abdomen was slightly full and tympanitic and some more characteristic spots appeared. The spleen had not been palpable at all up to this date. Constipation was the rule but on the tenth day there was one loose dark motion. His mind was quite clear, even acute; he slept well at night and never muttered or wandered during sleep. He began to look drawn but did not present the typhoid facies; he had no tendency to somnolence and watched narrowly everything which went on in his room. In the third week the temperature resembled that seen during the fourth week of an average case of enteric fever when it generally oscillates from 97° to 102° before finally settling down. The pulse dropped in rate and did not reach 100 beats per minute; it remained, however, very soft, almost collapsing. The heart remained normal though there was a tendency for the beats to approach each other in character. Spots continued to come out; the abdomen was less distended. The spleen for the first time was just palpable. Occasional diarrhoea of a light colour was noted, though enemata were generally needed. The other signs were those of declining enteric fever. On the nineteenth day he complained of pain in the left groin and down the leg, accurately following the course of the femoral vein. Belladonna and glycerine were applied and the limb was put on a Macintyre splint. This symptom became more marked during the next few days and there was undoubted thrombosis of the left femoral vein and the thigh was one and a half inches larger than its fellow and the calf of the leg one inch larger in circumference. He was drawn and thin in appearance but was not like a typical typhoid case. In the fourth week the temperature remained as before until the twenty-third day, when it kept up, and on the twenty-fifth day it shot up to 103·6° and at midnight to 106° and was accompanied by a rigor, followed by considerable collapse. The pulse was generally over 100 and was soft and collapsing. Towards the end of



A, sponging. B, rigor. C, quinine. The rigor on March 4th was "very small." The daily amounts of quinine were 40 grains on Feb. 2nd, 36 grains on the 3rd, 40 grains on the 4th, 27 grains on the 26th, 27 grains on the 27th, 39 grains on the 28th, 30 grains on March 1st, 40 grains on the 2nd, 40 grains on the 3rd, and 40 grains on the 4th.

but chills were felt occasionally and copious sweating, especially during sleep. The pulse varied from 90 to 110, was very soft, and was markedly dicrotic from the first. The tongue was covered with thick white fur up to the edges. The abdomen was normal at first but on the sixth day was slightly full but not tender. Gurgling in the right iliac fossa was noted on the third day and persisted. One spot was seen in the right iliac region on the sixth day. He slept well but on one or two nights took ten grains of Dover's powder. There was a tendency to constipation during the first week. Calomel was given on the second day and enemata afterwards. The headache persisted and was severe up to the seventh day and then began to decline. Quinine was given for the first four days but as it did not affect the temperature it was then stopped. The usual sudorific mixture was given. The diet was milk and soda. In the second week the temperature took a higher level and varied from 101·8° in the morning to 103·8° in the evening. On the morning of the 14th it dropped to normal for a few hours. The pulse varied between 82 and 104, the dicrotism was less marked, and the

the week the first mitral sound became almost inaudible and the sounds over the other regions were embryonic in character. There was no access of pain in the thigh at the time of the rigor. The tongue was patched with thin brown fur. The abdomen was flat but more spots were erupted at intervals during this week. There were a pleuritic pain low down on the left side and a slight irritating cough and discharge of a small quantity of glutinous phlegm. Auscultation revealed nothing. The leg continued to give trouble and definite resistance and pain were felt on tracing down the course of the vein. The right thigh was 19½ inches in circumference and the left thigh was 20½ inches. During the week cardiac stimulants were given, including brandy, three ounces per diem, and a mixture containing ammonia, digitalis, and strychnine. Quinine was given as follows: on the twenty-sixth day, 27 grains; on the twenty-seventh day, 27 grains; and on the twenty-eighth day, 39 grains. The temperature was not affected. Tepid sponging was given when the temperature reached 102·5°. The patient was drawn but his mind was clear and he slept well. In the fifth week (March 1st to 7th),

with regard to the temperature, rigors occurred on March 1st and 2nd respectively, the temperature reaching each time 105°. Heavy sweats followed and some collapse requiring stimulants. Otherwise the patient was more cheerful and better. The leg was less swollen and the pain was less. The abdomen was flat, the tongue was still patchy, and there was occasional diarrhoea of light stools. The patient continued to take about three pints of milk daily and broths. Bacilluria was noted and urotropine in eight-grain doses was given twice daily. The heart was weak and the brandy was therefore increased to four ounces daily. On the last day of the fifth week injections of quinine were commenced to see if the hitherto erratic temperature could be controlled. In the sixth week about 15 grains of sulphate of quinine were injected by the hypodermic method into the skin of the arm every day. The injections gave rise to no trouble except transient irritation and redness and subsequent induration of the sites. All the symptoms declined gradually and hereafter nothing of interest occurred. The pulse recovered its tone slowly. The lungs were examined at intervals and were normal throughout. The urine was examined four or five times; the specific gravity was generally about 1020 and no albumin or sugar was ever present. The diazo reaction was not examined for, owing to lack of the necessary reagents. Indican was abundant in the third week and almost non-existent subsequently. Convalescence was tedious and the officer was not in a fit state to travel home on sick leave until April 29th, nearly 12 weeks from the date of his attack.

The condition of the blood was as follows. On Feb. 3rd (the third day of the disease) the leucocytes were 6250 per cubic millimetre. Differential count (400 cells counted): polymorphonuclears, 60 per cent.; small mononuclears, 28 per cent.; large mononuclears, 11 per cent.; eosinophiles, 0; and mast cells, 1 per cent. No malarial organisms were seen after careful examination. There were no pigmented leucocytes. On Feb. 19th (the nineteenth day of the disease and the one on which femoral thrombosis occurred) the condition of the blood was as follows: hæmoglobin, 100 per cent.; red cells, 5,000,000 per cubic millimetre; white cells, 8125 per cubic millimetre; individual corpuscular richness, 1. Differential count (400 counted): polymorphonuclears, 72 per cent.; small mononuclears, 20 per cent.; large mononuclears, 7 per cent.; eosinophiles, 1 per cent.; and mast cells, 0. No parasites were seen. No pigmented leucocytes or debris of parasites were seen. On Feb. 25th, the twenty-fifth day of the disease (the day of the rigor and high rise of temperature), the white cells (average of two counts) were 9787 per cubic millimetre (400 counted); polymorphonuclears, 74.5 per cent.; small mononuclears, 17.0 per cent.; large mononuclears, 4.5 per cent.; eosinophiles, 2.0 per cent.; finely granular basophiles, 1.5 per cent.; and coarsely granular (mast cells), 0.5 per cent. The blood film was normal. No organisms were seen. (It was carefully searched on several subsequent occasions and no adventitious bodies were found.) On March 2nd, the thirty-first day of the disease (during the rigor week), the blood condition was as follows: hæmoglobin, 96 per cent.; red blood corpuscles, 3,513,000 per cubic millimetre; white blood corpuscles, 10,312 per cubic millimetre; and individual corpuscular richness, 1.37. Differential count (300 cells counted): polymorphonuclears, 76.6 per cent.; small mononuclears, 10.7 per cent.; large mononuclears, 10.4 per cent.; eosinophiles, 2.0 per cent.; and mast cells, 0.3 per cent. Three nucleated red cells (normoblasts) were counted. A few doubtful intracellular bodies were seen but they did not react to Romanowsky's stain in the characteristic manner of malarial parasites. No pigmented leucocytes were seen. On April 10th during convalescence the condition of the blood was as follows (the average of three observations was taken during the same hour): hæmoglobin, 100 per cent.; red blood corpuscles, 5,083,000 per cubic millimetre; and white blood corpuscles, 10,312 per cubic millimetre. The fresh blood was carefully examined without any result. The differential count (300 examined) was as follows: polymorphonuclears, 32.3 per cent.; small mononuclears, 35.3 per cent.; large mononuclears, 3.2 per cent.; eosinophiles, 28.6 per cent.; and mast cells, 0.6 per cent. These notes were made at the time. All the cells were perfectly distinct and characteristic and the slide was clear and well stained (Romanowsky). The protoplasm of the polynuclear cells was almost hyaline or at the most very faintly granular. The small mononuclears had a fair circle of protoplasm

around the nucleus and were quite typical. The large mononuclears were very few in number but were quite typical. The extraordinary number of eosinophile cells was a very striking feature. They were ordinary coarsely granular eosinophile cells with a multiple nucleus but the limiting cell membrane was more defined than often is the case and there were none ruptured during the process of making the film. No parasites were seen and there were no pigmented leucocytes or malarial debris.¹ On April 20th the red blood corpuscles were 4,666,600 per cubic millimetre and the white blood corpuscles were 9375 per cubic millimetre. Seen in the fresh state large numbers of coarsely granular cells were seen. The red blood corpuscles formed rouleaux well. No parasites were seen. There was no movement of the red blood corpuscles or in the plasma in a hanging drop. The evening blood showed no filaria. The differential count of the above was as follows (200 counted): polymorphonuclears, 47.0 per cent.; small mononuclears, 12.5 per cent.; large mononuclears, 5.5 per cent.; eosinophiles, 35.0 per cent.; and mast cells, 0. The cells again were all just as in the previous blood of April 10th, but the eosinophile granules took on a less decided red and a more neutrophile reaction. (This, I believe, was due to the staining process, because the red blood cells in the former slide were pink and on this slide green. In other words, the second stain used was deficient in eosine.)

Widal's examination.—The blood was first sent up to the Pasteur Institute at Kasauli and after examination Captain G. Lamb, I.M.S., reported: "No reaction with 1 in 40 dilution." Another strain of bacillus typhosus was then obtained from Captain Kelly, I.M.S., of Shansi, a strain which had originally come from the Medical College, Calcutta. I made dilution of 1 in 2, 1 in 20, 1 in 100, and 1 in 200, and the following was noted. After 15 minutes a slight reaction was noted in the 1 in 2 and in the 1 in 20 dilution. This reaction was not increased at the end of an hour and at that time only a few small clumps were seen and the large majority of the bacilli remained actively motile. At the end of an hour no perceptible reaction could be observed in the 1 in 100 or in the 1 in 200. Latterly another strain of bacillus typhosus was obtained from the Kasauli Institute. Again no reaction whatever was noted after one hour in dilutions of 1 in 20, 1 in 50, and 1 in 100.² Cover-glass preparations made from the sputum showed a few coccoid bacilli mostly in groups or clumps. A few elements were longer and evenly stained but most were segmented and showed polar staining. No true cocci were present. Unfortunately, the lack of any apparatus in such an out-of-the-way station prevented any attempt being made to isolate the causative organism bacteriologically.

Remarks.—I think there is good ground for believing this to be a case of paratyphoid fever, inasmuch as clinically the disease ran a course very like true enteric fever and yet the serum failed to give the characteristic reaction with no less than three different strains of the Eberth-Gaffky bacillus. Although the clinical course of the disease was one which might stand for a typical attack of typhoid fever, yet on closer examination there were several features so unusual as to call for special notice. In speaking of the clinical picture presented by "the typhoid state" its value as a symptom depends entirely on the experience the individual observer has had in the normal course of the disease. To experienced practitioners the diagnostic value of the "typhoid state" transcends all other individual symptoms in importance and it was just this important symptom that was completely absent in the case under consideration. Previously to coming to India I had a series of nearly 300 cases under my immediate care spread over several years and so the clinical picture of British typhoid fever was a familiar one and during the unfolding of this case of fever the absence of all mental symptoms of true enteric fever struck me as most anomalous. Instead of the characteristic somnolence and apathy there were a hyperacuity, an almost morbid inquisitiveness, and an apprehensive watching of the most trivial details of nursing. Again, the course of the temperature was abnormal. The wide diurnal variation within a more or less limited range is suggestive of a septic rather than a typhoid process. This

¹ Santonin was given at night in full doses and sulphate of magnesia in the morning. Free purgation resulted but no worms were found. This was repeated with thymol without effect.

² On another occasion some blood was sent to the Petit Laboratory in Bombay, there to be tested with Ehrlich's original cultures of Eberth's bacillus, Gaertner's bacillus, and the bacillus coli communis. Unfortunately, owing to incomplete sealing of the tubes, the serum was not in a fit state for examination on arrival.

type is seen during the fourth week of an average typhoid case and corresponds to the time when the ulcers are bare and absorbing small quantities of septic material. In the case under consideration this septic type persisted from the beginning and in the fourth week the high rises and sudden falls and the rigors and sweats suggested either malarial fever or pyæmia. The former was disproved by blood examination and there was no reason to suppose that true pyæmia was ever present. The abdominal symptoms were less marked than usually occurs in a case of such severity. It is true gurgling in the right iliac fossa was marked from the first but the abdomen was never more than slightly full and the spleen was not enlarged at any time. The tongue never became dry and sordes never formed. The case was a sporadic one; there was no enteric fever at Lucknow, and 25 days had elapsed since he left that station before the first symptom occurred. No case had ever occurred in Goona in the memory of anyone. He may have picked up the infection from a railway refreshment room *en route*.

The condition of the blood.—In true enteric fever the following changes are well recognised. 1. The red corpuscles are progressively reduced in number until when convalescence is established and post-typhoid anæmia is present the number may have sunk to half the normal. This, as Hayem has pointed out,³ may be modified at any time in the course of the disease by watery discharges acting by thickening the blood and producing a spurious polycythæmia. 2. The white corpuscles are unaffected or more often reduced in number. A marked hypo-leucocytosis is frequent but a true leucocytosis is said by some (von Limbeck and others) to supervene with the advent of certain complications, an observation, however, denied by many. I have examined several cases presenting complications, such as pneumonia, cystitis, and laryngitis and periostitis, and in none of these was there leucocytosis. Many of the complications of typhoid fever are due to the typhoid bacillus, but some are complicated by the occurrence of the common organisms of supuration; this is the probable cause of the discrepancy. The complications caused by the pure typhoid bacillus show no leucocytosis but with the advent of cocci comes a rise in the leucocyte rate. As the disease progresses the polynuclear leucocytes diminish in greater ratio than the mononuclear, till late in the disease the latter exceed the former. In the case under observation the general characters are those of true enteric fever, but the leucocyte count was higher than is usual in enteric fever. This suggests, like the temperature chart, a septic element or at any rate a mixed typhoid infection.

The red blood corpuscles were never much reduced and on April 10th the anomaly of a polycythæmia in an obviously anæmic patient was seen. The condition of his mucous membrane suggested a moderate though marked degree of anæmia and yet three successive blood counts gave an average of over 5,000,000 per cubic millimetre. It is a good instance of the disturbance of the other element in the blood count, the degree of plasma dilution, an element too frequently overlooked in making a decision on blood count. The large mononuclear leucocytes were somewhat high until convalescence. This is a very important factor in the diagnosis of malarial fever, as Christophers and Stevens showed, but in such cases the percentage is generally 16, 20, or more. The great increase of eosinophile cells in the latter examination is a very curious feature and the most obvious suggestion that it was caused by worm parasites was disproved. I saw a case of Kanthack's which showed 62 per cent. of eosinophile cells in the circulating blood of a man with general pemphigus. This increase has been noticed since in similar cases and the bases of pemphigus blebs will generally be found paved with eosinophile cells. In the subcutaneous tissues of the gut in acute dysentery I have noticed masses of the same cells and in the blood of patients with scarlet fever and mania. The phenomenon is generally stated to be marked in cases of asthma. In two cases seen by me during the last month, one, a case of limited pemphigus, showed 43 per cent. of eosinophile cells, and the second, an inflammatory condition of the calf of the leg around an encysted guinea-worm, the percentage of these cells was 35.

An interesting feature common to all these conditions is the involvement of extensive areas of subepithelial connective tissue (many intestinal worms, especially ankylostomata, produce this change), and Kanthack and Hardy

were of opinion that eosinophile cells were derived from the body connective tissue. The relation between these two conditions, though purely hypothetical, is suggestive. The total evidence of the blood state was that the disease was enteric fever or something very like it.

Widal's test.—The absence of this reaction with three distinct strains of the Eberth bacillus may be said to prove that in the case in question the *causa causans* was not the Eberth-Gaffky bacillus but some other. A positive reaction would, indeed, have had less value, for even if the Eberth bacillus were playing a subsidiary part in the infection then a positive reaction would have been present but the total absence of any agglutination is strong evidence of the total absence of the usual bacillus of enteric fever. The blood was sent for examination with the Gärtner bacillus and the bacillus coli but, as before stated, it met with an accident. Of the great value of the Widal-Grünbaum reaction there is no question but it is certain that it is not so immaculate as its devisers first claimed. "This is enforced by the fact, which appears to be established by Jürgens Brion and Altschüler and Stern in their studies of the agglutination curves during the course of the disease, that maximum agglutination value may be given by the serum with bacilli which were shown to have no etiological relation to the illness and, further, that the specific agglutination value is not invariably above that of the group agglutination, which, indeed, cuts at the root of the *rationale* of the differentiation of paratyphoid and enteric fevers."⁴ If this were generally the case then the possibility of separating the various members of the typhoid-Gärtner-coli family would cease but, speaking generally, it is possible to come to an accurate conclusion by the reaction of the various members in increasing degrees of dilution.

That organism which continues to be agglutinated by the serum in highest degrees of dilution is almost certainly that most intimately connected with the disease produced. Thus the writer in the same report says:⁵ "The weight of evidence, which is abundant and daily accumulating, goes to show that while paratyphoid serum will often agglutinate bacillus typhosus it always acts upon the paratyphoid bacillus which is in causal connexion with the attack in very much higher dilutions. The pathological results of infection are as a rule conformable to the clinical effects—that is to say, the picture may frequently stand for an ordinary case of true enteric but as a rule there are modifications due to the absence of one or other typical feature of the latter." Again, Major W. H. Horrocks⁶ writes: "Schottmüller has described a number of cases of 'paratyphus.' The symptoms re-embled very closely those seen in true cases of enteric fever. The temperature charts were so characteristic that the diagnosis of enteric fever seemed almost beyond cavil. Rose spots, enlargement of the spleen, and diarrhoea were also observed in some of the cases but in others the abdominal symptoms were not so marked. When tested with the serum from an undoubted case of enteric fever the bacilli showed no traces of agglutination but when tested with serum collected from each of the cases all the bacilli became agglutinated. The reaction was observed not only with the bacilli and serum from the same case but also between the bacilli and sera of all the cases. The effective dilutions of the sera varied from 1-100 to 1-10,000. The bacteriological results obtained seem to prove conclusively that the cases of 'paratyphus' were caused by a variety of the bacillus of Gärtner."

Dr. R. Tanner Hewlett, in an article in the *Journal of State Medicine* for November, 1903, describes some cases where the symptoms clinically were those of true enteric fever but where the serum failed to agglutinate cultures of the typhoid bacillus. Several of these were found to be associated with the bacillus enteritidis or Gärtner group and in several cases this bacillus was cultivated in a pure state from the body and the serum had high agglutinating powers on the same organisms. More conclusive proof still is afforded by an interesting and valuable paper by Dr. R. Row of the Petit Laboratory, Bombay, entitled "Obscure Irregular Continued Fevers of the Typhoid Group and their Probable Relation with Different Species of Bacilli of the Typho-Coli Race, as seen from the Specific Bacteriolytic Value of Blood Serums of such Cases on various Members of the Typho-Coli Group of

⁴ Annual Report of the Sanitary Commissioner with the Government of India for 1903, p. 20.

⁵ Ibid., p. 19.

⁶ Bacteriological Examination of Water, 1901, Churchill.

³ Johns Hopkins Hospital Reports, vol. iv.

Microbe." The method which this observer adopted was as follows. Equal parts of the fresh serum of the patient under examination and of young typhoid, coli, or Gärtner cultures were put up in the form of hanging drops under aseptic conditions. These were examined as fresh specimens and subsequently at determined intervals were removed, dried, and stained in the usual way. Others were allowed to stand for 24 hours and subsequently inoculated on to agar and the degree of growth noted. In all, records of 24 cases are given and the majority of these were examined with the three types of organism above mentioned. As a result, all degrees of reaction were demonstrated; some serums reacted to all three organisms well, more reacted to one intensely and the other two feebly, and some reacted to one strain only and not to either of the others—i.e., suggested pure infection. If such a deduction is justifiable it shows that in almost all the cases brought forward there was mixed infection; some cases of typhoid fever were caused by the Eberth bacillus, some cases of typhoid fever were caused either by the Gärtner bacillus or by the bacillus coli, and more were associated with both or all three bacilli.

The reactions produced *in vitro* in the above experiments were one or other of the following: (1) complete bacteriolysis; (2) partial bacteriolysis; (3) definite agglutination (produced by agglutin already present in the serum or subsequently produced *in vitro* by the leucocytes, some of which are accidentally carried into the serum); (4) indefinite agglutination; and (5) a negative reaction.

Without going into further details Dr. Row's conclusions are as follows (some of lesser importance are omitted): 1. The blood serum of patients suffering from continued fevers under consideration acts powerfully on the bacilli of typho-coli race and the serum reaction appears to be specific towards one or other members of the group. 2. The reaction is of a lysogenic nature. 3. Whilst some of the serum reactions are referable to a pure bacillus typhosus infection a good number of them point to infection by organisms other than the bacillus typhosus—viz. bacillus enteritidis Gärtner and bacillus coli communis. 4. Shows that the reaction is sometimes pure but at others is focal for one organism and more diffused for the other members of the group. 5. The effort of nature in producing anti-bodies specialises as the case progresses, the earlier process being a sort of general reaction. 6. Relapse in enteric fever proper may be caused by coli infection superimposed on true typhoid fever. 7. Pure bacillus coli infection seems to be the most common cause of atypical and irregular fevers dealt with here. However, it is not unusual to meet with infection by bacillus enteritidis Gärtner, or mixed infection of two or more groups of organisms of this class. He concludes as follows. 8. These considerations seem to support my previous observation on the close relationship of the different members of the typho-coli race—the products of this group of organisms (as I then experimentally proved) when administered intravenously into rabbits inducing a train of closely allied symptoms and also marked intestinal lesions of a similar nature.

Paratyphoid fever in India.—Attention of medical officers in India is drawn to this subject in the Sanitary Commissioner's blue-book for the year 1902 above mentioned but in

the article no special reference to the disease in India is made. In the same blue-book for 1903 an excellent *résumé* is given of the subject but again no special mention of the Indian atypical typhoid cases is made. At the same time it will probably be found that these irregular manifestations are particularly common in India, at any rate as compared with England. My personal experiences with clinical enteric fever in England and the Widal reaction showed a failure of less than 4 per cent. and if allowance be made for personal and technical failures it is still less. The 24 cases collected by Dr. Row, the experiences of Lieutenant-Colonel D. B. Spencer, I.M.S. (to be hereafter related), my present case, and others go to show that paratyphoid fever is quite common in India. It is very desirable to know to what extent it really occurs and it is only by careful observation of individual cases and repeated Widal examinations that the condition can be detected. The evidence of post-mortem examinations cannot be over-rated and it is possible that the type, position, distribution, or character of the intestinal ulceration may be different. From time to time puzzling accounts have been published where cases clinically diagnosed as enteric fever and giving Widal reaction were found at the post-mortem examination to present no intestinal ulceration. These may have been cases of paratyphoid fever, for admittedly the abdominal signs in such are usually less than in true enteric fever and presumably, therefore, the ulcers were also less severe.

A very tempting subject is that of the resemblance between enteric fever, paratyphoid fever, and dysentery, and the affinities between the Eberth-Gaffky bacillus, the coli communis bacillus, and Shiga's bacillus of dysentery. It might be shown that there is a suggestive gradation between the types, not only in their clinical manifestations, the site and type of the intestinal ulceration, and the degree of septicity of the fever, and so on, but more markedly in the biological gradation from the bacillus at one end of the arc to that at the other end, but the subject is scarcely ripe for discussion and it only needs to be borne in mind in order to show the importance, difficulty, and complexity of the whole question.

In the *Indian Medical Gazette* for April, 1900, there appears a paper entitled Enteric Fever in India, by Lieutenant-Colonel (then Major) Spencer, I.M.S. This appears to be the first paper definitely calling attention to the question of atypical typhoid fever in India and in it Lieutenant-Colonel Spencer challenges the unity of enteric fever. To the paper Dr. Row from the bacteriological side makes a fitting pendant and practically proves the whole question. Lieutenant-Colonel Spencer's paper is a long one and it is sufficient to refer briefly to a few of his most important points. He notes: (1) the clinical differences (*vide table*); (2) the rarity with which the typhoid bacillus is ever detected in food or water and its frequent absence in fæces or internal organs of those cases examined with this view (in India); (3) irregularity of the Widal reaction; and (4) that though for years the greatest care has been taken to prevent dissemination of infection in Indian cantonments yet enteric fever continues to be frequent. Many Indian cantonments are models of scientific sanitation but enteric fever seems to be almost unaffected.

* TABLE I.—*True Enterica.*

Etiology.	Widal test.	Mode of incidence.	Mortality per cent.	Chart.	Rash.	Typhoid state.	Abdominal symptoms.	Post-mortem appearances.	Duration of fever.	Treatment.
Bacillus typhi (Eberth) associated with sewage contamination of food or drink.	Positive reaction with the bacillus typhi.	Usually in epidemic form.	7-14 per cent. Maidstone epidemic (Poole).	Often typical; first week, gradual rise; second week, high continued fever; third week, gradual defer- vescence.	Generally present. 88 per cent. in Maidstone epidemic (Poole).	Early and pronounced.	Whether early or late they are unmis- takeable.	Typical ulceration of glandular structures of small intes- tine.	Generally three weeks.	Expectant.

TABLE II.—*Indian Enterica (a Fever with Enteric Symptoms).*

Probably the bacillus coli associated with fermentation and putrefac- tion of intes- tinal contents and consequent auto-infection.	Negative.	Usually sporadic.	About 25 per cent. in India.	Generally irregular.	Generally absent.	Often vague or alto- gether absent.	Often absent.	Often the ulceration is irregular and extensive, being uncon- fined to glandu- lar structures.	Three or four weeks but can be aborted by a special treat- ment.	The treat- ment I adopt is eliminative, combined with intes- tinal anti- sepsis and irrigation.
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* Lieutenant Colonel Spencer's chart from the Indian Medical Gazette for April, 1900 (by permission).

After showing the ubiquity of the bacillus coli communis as an intestinal saprophyte, I must state my firm belief in auto-intoxication by that organism and explain the whole question by reference to certain unknown conditions of lowered body resistance culminating in what amounts to transmutation of a saprophyte into a parasite. Ptomaine poisoning may be regarded as an evidence of a similar process and therefore allied to a typical typhoid or paratyphoid fever, as may be seen from Lieutenant-Colonel Spencer's interesting comparative charts. In a private communication Lieutenant-Colonel Spencer points out that he uses an "abortive treatment" in these cases which consists in frequent flushing of the colon by large enemata of Condry's fluid and the administration of sulphate of quinine and sodium salicylate in alternate doses. He writes: "By this means I have never failed to bring the case to a favourable termination by the end of the second week." I have for several years been an advocate of colic lavage (izal) in dysenteric conditions⁷ but where the ulceration is above the ileo-cæcal valve this treatment would appear to be of little value.

Granting, then, that paratyphoid fever exists and that in India it appears to be commoner than in England one or two important questions arise which certainly cannot be answered at present. Is there, after all, a transmutation of different species of bacilli as was once claimed in the case of the bacillus anthracis and the hay bacillus? How does the bacillus coli communis attain its power of changing its saprophytic habits for parasitic ones? Is it as a result of lowered resistance of its host or certain unknown changes in man which allow auto-infection or is the power gained by the bacillus in its extra corporeal existence? The conditions of life vary so much in the tropics from those of the temperate zone that it is idle to seize upon one factor. Nevertheless, one may suggest the frequency with which human faeces pass through the bodies of other animals as a possible cause.

In England none of the higher animals feed on human excrement, but in India there are many—thus the pig, the dog, and certain carrion birds do so habitually—i.e., it is their staple article of food. Hares, partridges, peafowl, cattle, and buffaloes are very frequently foul feeders and any or all these latter are eaten by man or in the case of cattle their milk is consumed. It is a matter of common knowledge that many organisms undergo an exaltation in virulence by being passed through the bodies of animals and it is possible that in this way increased potentialities for evil are acquired by the typho-coli group. Enough has been written to show that another problem enters into Indian sanitation, the origin of paratyphoid variations. Are we, therefore, to go on looking upon the bacillus coli communis and its congeners as harmless organisms to be tolerated in pure water? Will it be necessary to inoculate our Indian drafts of soldiers with a polyvalent antityphoid vaccine like that being made polyvalent for the various micrococci of disease? Whatever answers will in the near future be given to these questions, one thing stands certain—the unity of typhoid fever can no longer be upheld.

My thanks are due to Dr. Row of Bombay for a demonstration of his specimens, methods, and literature, and to Lieutenant-Colonel Spencer for much help and the use of his published articles.

Bombay.

INFECTIVE PURPURA.

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SEVERAL considerations have prompted me to place the following case of purpura on record. The remarkable features and the extreme gravity of the condition from which recovery eventually took place are in themselves sufficient justification for this, especially as reference to the literature on the subject shows our present knowledge to be very imperfect. Furthermore, I am anxious to call attention to the beneficial influence which the prolonged administration of quinine exerted on the clinical course of the illness,

for in regard to treatment also literature leaves much to be desired. The following is a short account of the case.

The patient, a married woman, aged 34 years, had been in a poor state of health for several months, but had had no definite illness until about 36 hours before my first visit. The symptoms complained of were severe frontal headache and pains in the back and limbs. It was evening when she was first seen and the temperature was 104° F. and the pulse was 124. A few small petechial extravasations were noticed and these were confined to the areas just above and below the knees. Six grains of salicylate of quinine and 15 grains of phenacetin divided into three cachets were given during the night with the result that the pains were eased and a rapid fall of temperature ensued, but this was accompanied by extreme restlessness and frequent attacks of vomiting. On the following morning the temperature was found to be subnormal but the pulse was still rapid (120). The signs of purpura were much more obvious and the area invaded became gradually extended until the following day when the limit appeared to be reached. The purpuric patches varied in size, the largest being situated in front of the knees and about three and a half inches in diameter. There were large patches in the skin over each elbow, on the dorsal surface of the hands and of the joints of all the fingers but not the thumbs. The feet were similarly affected but to a lesser extent, the skin over the malleoli and the metacarpo-phalangeal joint of the great toes showing the largest patches. Similar extravasations were also noticed behind the spines of the scapulæ and on the lower part of the back. There were numerous smaller patches on the dorsal surface of the limbs, the largest of these being nearest to the elbows and knees. The distribution suggested that the amount of extravasation was greater in the area of skin stretched over bony prominences and was promoted by the retarded circulation in these parts. The mucous membrane was not affected so seriously as the skin. There was slight hæmorrhage from the mouth; and the surface of the lips, mouth, and tongue was covered with a yellowish white slough and the mucous membrane of the nostrils and conjunctivæ was similarly affected. There was also a slight bloodstained discharge from the other mucous membranes, but during the whole course of the illness there did not appear to be any sign of internal hæmorrhage and the retina was also quite free. The area of the various patches had reached its maximum within 36 hours of the first appearance of purpura and no further extravasations took place. The urine was examined at this stage and was found to contain albumin—about one-sixth. The patient continued to be very restless and collapsed and could not be induced to take even liquid food by the mouth. Rectal feeding was employed and to the nutrient enemata salicylate of quinine and brandy were added. The restlessness continued and it was only after successive doses of opium—trional and ammonium bromide having been found useless—that any sleep was obtained. About the fifth day the mucous membranes of the mouth, tongue, and nostrils began to slough and respiration was greatly impeded. The nose and throat were frequently washed with boric lotion by means of a nasal douche and the fauces were painted with glycerine of borax. In the course of a few days necrotic patches of mucous membrane began to separate but there was no deep sloughing of these parts. The purpuric patches scattered over the trunk and limbs pursued the following course. The superficial layer of the skin became dry and the margin raised by an exudation at first serous and then purulent. In the smaller patches the skin could be peeled off in course of time, leaving a slightly pigmented surface. In the case of the larger patches, on the removal of the skin a smooth surface, dark brown in colour, was exposed. At first this was quite dry but soon pus began to exude round the edges and when this necrotic portion became separated the tissue beneath was that of a sloughing sore. The depth of each slough varied directly with the size of the original patch. Thus, in the case of the elbows and knees the periosteum was exposed to a considerable extent, and on the dorsum of the hand the sheaths of the extensor tendons were clearly visible. The back of each hand was practically one large slough. The inner surface of the great toes showed a similar condition. When the disease had lasted for about a month these sloughing surfaces were at their worst. They became gradually more healthy and began to granulate, and at the end of three months they were all soundly healed. During the period when the sloughing was very extensive—for

⁷ Vide the Special Dysentery Number of the Indian Medical Gazette, July, 1905.