discharge with the drainage tube still in. He was admitted once more in December, 1903, looking very ill and thin. His general health soon improved, but the old sinus showed no signs of closing. On June 16th, 1904, the cavity was curetted and washed out with an iodine solution. In August the wound began to heal, though still discharging freely. The patient had cavities at both apices of the lungs; he frequently coughed up sputa, which, however, contained no tubercle bacilli. The liver appeared to be enlarged (commencing amyloid disease?). His general health had not improved as on former occasions, though he gained slightly in weight; his heart was drawn to the left side of his chest. The sinus showed no signs of healing, his night sweats increased, and there was a rise of temperature every evening. He was anxious that something more should be done, and on February 1st, 1905, after explaining the danger of the operation to him, I decided to excise some ribs and give the man another chance of life. Every necessary precaution was taken before the operation. Brandy was given by the mouth and strychnine injected; he was clad in flannel and reated on hot-water pillows during the operation. Chloroform was first given, followed by A.C.E. mixture; the old discharging sinus was slit up, to allow the cavity to be well explored with the finger; the cavity was found to extend as far as the sixth rib; the pleura

The shaded portions show the ribs removed during the thoracoplasty; the non-shaded portion in the ninth rib represents the first piece of rib excised soon after admission. A, Vomicae; B, U shaped flap corresponding to the cavity.

was much thickened. A large U-shaped flap, corresponding to the cavity to be closed, was made; the flap was then raised and turned upwards, including skin, fat, and muscles. The incision of the flap commenced from the middle of the vertebral border of the scapula on the level of the sixth rib (see diagram). This extended downwards to the upper surface of the tenth rib, below the discharging sinus, finishing at the axillary border (see diagram). The ribs with their intercostal muscles were exposed and were removed after being shelled out of their coverings, dividing them anteriorly and posteriorly. About 3 or 4 in. of the seventh, eighth, and ninth ribs were removed. The remaining soft parts, which consisted of muscles, thickened pleura, and periosteum of the ribs, were cut away with scissors. The bleeding was not excessive, but a few vessels were tied. The visceral layer of the pleura was then exposed. It was covered with prominent, unhealthy granulations; these were freely curetted, and pressure was applied by means of swabs soaked in adrenalin. The patient at this stage suddenly collapsed, and the operation was finished as speedily as possible. The U-shaped flap of skin and muscles was brought down to cover the visceral pleura; the flaps were stitched together, the old sinus in the lower part of the flap was cut out, and a drainage tube inserted. The wound was hurriedly closed and the patient put quickly to bed and a saline injection given, after which he soon rallied.

The man's general health and condition rapidly improved; he gained 3 st. in weight, and was discharged July, 1906 looking fat and well.

A TYPHOID CARRIER OF TWENTY-NINE YEARS' STANDING.

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THE following case seems worthy of record from its almost unique character, and it serves to illustrate and elucidate a number of points in connexion with recent advances in our knowledge of the pathology and etiology of enteric fever.

History of the Case.

Twenty-nine years ago Dr. W. T., a medical practitioner had as a young man, during a residence in the United States, a severe attack of typhoid fever. During the hungry stage of his convalescence, he suffered from dyspepsia on several occasions, and within three months of the period of his illness, he had an attack of very acute pain in the region of the liver, with a feeling of distension in the neighbourhood of the gall bladder.

Attacks of this character since recognized by him as biliary colic recurred about every three months for over a year. Since that time they have become less frequent, but every year one or more attacks occur, often during holidays, and are generally associated with over exertion, long bicycle rides or walks, and irregular meals. Jaundice has sometimes been a feature of these attacks. His bowels act regularly, and his evacuations are normal. With the exception of these attacks, his health has been otherwise excellent, and he has carried on a very heavy practice during the last twenty years without being a single whole day off duty through ill-health. In view of the recent work on typhoid fever, it occurred to me that the case might be one of cholecystitis typhosa, and that W. T. was possibly a typhoid carrier. On discussing the question with him, he readily put himself at my disposal for investigation.

Bacteriological Examination.

In selecting the samples for plating, any mucus present in the faeces was included. A platinum loopful of faeces was emulsified in a broth tube containing 5 c.cm. of broth. Three loopfuls were then placed on the first of a series of Petri's plates containing MacConkey's bile-salt-lactose-neutral-red-agar. By means of a glass rod the material was distributed over the surface of the medium, and the same rod was employed for the inoculation of the second and third plates of the series.

Three samples have been examined, and in all of them the typhoid bacillus has been demonstrated. The first sample gave only non-lactose fermenting colonies, all of which proved to be *B. typhosus*. The second sample gave about equal numbers of colonies of the typhoid bacillus and of *B. coli communis*, and in this case a few colonies of the *Bacillus faecalis alkaligenes* were also present. The third sample gave seventy colonies of the typhoid bacillus and one colony of *B. coli communis*.

That we were here dealing with the typhoid bacillus was shown as follows:

The bacillus is a highly motile rod; it does not liquefy gelatine and does not produce indol. It produces acid, without gas formation, in litmus-peptone-water media containing glucose, laevulose, galactose, maltose, dextrin, and mannite, and no change in arabinose, saccharose, lactose, raffinose, inulin, erythrite, and dulcitol; it produces acid in litmus milk.

The urine was examined but no typhoid bacilli were found. The bacillus is pathogenic for the guinea-pig. Three guinea-pigs were inoculated intraperitoneally with 0.5 c.cm., 1 c.cm., and 2 c.cm. respectively of a twenty-four hours' culture in broth. The guinea-pig which received 2 c.cm. died within twenty-four hours, the other two animals survived.

The bacillus is agglutinated by an antityphoid horse serum in a dilution of 1 in 10,000.

Agglutination Tests with Patient's Serum.

With the bacillus ordinarily used in the Institute for the Widal test the patient's serum gave an agglutination in a concentration of 1 in 25 but failed to do so in 1 in 50, and 1 in 100.

With the patient's own bacillus the patient's serum failed to produce agglutination even in 1 in 25. That the absence of this reaction with the patient's own serum is not due to the lack of suitable receptors in the bacillus is proved by the fact that it was capable of being agglutinated by immune horse serum.

Within the last two years a revision of our conception of many of the factors concerned in the epidemiology and pathogenesis of typhoid fever has become necessary, and this has been almost entirely due to the work of Förster and his collaborators in the Strassburg laboratories. That work is being confirmed on all sides at the present time. Formerly we pictured the typhoid bacillus as finding its way into the alimentary canal, multiplying there, and invading the tissues, chiefly the lymphoid tissues, of the small intestine and the spleen, and only secondarily and fairly late in the disease finding its way into the blood stream.

The new conception as outlined by Förster, is briefly as follows: The bacilli have been shown by Conradi to be present in the blood during the incubation period, and even before the bacilli can be found the presence of antibodies may be demonstrated by the precipitin test, as was shown by Fornet. On the other hand, it is only during the first and second weeks of the disease that the bacilli appear in the faeces. The bacilli, therefore, do not multiply in the gut, but at a very early stage find their way through the wall of the alimentary canal, perhaps even from the fauces and tonsils into the lymphatics, and thence into the blood. While the bacillus is producing its effects on the lymphoid tissues of the gut, etc., other and not less important events, which till recently had been overlooked, are taking place.

At a very early stage the bacilli find their way into the liver and bile, and here their presence gives rise to inflammatory changes in the bile ducts and gall bladder. These changes have been observed in cases of disease in man, and also after the intravenous injection of the microbes into animals. The normal bile, according to Förster, is not a very suitable medium for the typhoid bacillus, but when albuminous material is added to it the bacillus finds in it an excellent pabulum. In the case of typhoid fever the albuminous material is supplied by the inflammatory exudate from the ducts and gall bladder, and the bacilli grow freely under these conditions in the gall bladder and pass out through the bile duct into the small intestine, thence into the large intestine, and are thrown out in the faeces. The irregular occurrence of the bacillus in the faeces is thus explained. In the later stages the bacilli from the ulcerated gut are added to those coming from the gall bladder.

In most cases recovery is accompanied by a cessation or clearing up of the inflammatory processes in the liver, bile ducts, and gall bladder; but in a certain number, about 2 per cent, of the cases this cholecystitis typhosa becomes a chronic process. Such cases are those which constitute the group of typhoid carriers. The bacillus continues to multiply in the gall bladder for periods varying from months to years, and to be thrown out into the gut and thence into the outer world. The gall bladder of such cases may be regarded as a normal habitat of the typhoid bacillus.

In certain of these cases gall stones occur, and it is still an undecided point whether these precede the typhoid fever, in which case they would play a part in the production of the typhoid carrier, or, as seems more probable, are the result of the processes indicated.

It is interesting to note that just as three-fourths of the cases of biliary calculi occur in women, so three-fourths of the typhoid carriers are also women.

That such typhoid carriers are, especially under certain conditions, a public menace has been proved by the investigation of many outbreaks. When the carriers are cooks, bakers, or in any way connected with the food supply, it has been conclusively demonstrated that they may act as the distributors of infection to many individuals. The importance of this question of typhoid carriers from the point of view of preventive medicine can hardly be overestimated. Such cases probably account for the sporadic cases the origin of which has, in the absence of this knowledge, been so puzzling. It also gives a key to the understanding of outbreaks of enteric fever among soldiers in the field. It is obvious that the first step in dealing with such cases is their recognition. This might be arrived at by systematic examination of the stools of all convalescents, and the knowledge of being a source of danger to others would in the case of many lead to greater care of the personal hygiene. I do not propose to enter into the discussion of this side of the question, as it has already been dealt with in this JOURNAL in a communication by Drs. A. and J. C. G. Ledingham.¹

We have in this case, therefore, a typhoid carrier, and everything in the history points to the conclusion that the condition had its origin in the attack of typhoid fever which occurred in America twenty-nine years ago.

The suspicion that this was a typhoid carrier was not aroused by the occurrence of cases associated with the carrier, as has been the case in most of the isolated carriers hitherto investigated, but was deduced from the history, because, so far as Dr. T. knows, not a single case of infection originating from him has occurred. The fact

that he is a bachelor with a small household has probably some influence in this, but it is counterbalanced by the fact that he has frequently lived in households containing from six to twelve individuals. This points to the conclusion that with educated people in fairly hygienic surroundings, and where personal hygiene is carefully attended to, the risk of infecting others is not so great as might be imagined. The very definite history of the first attack of biliary colic in a young man occurring three months after typhoid fever, strongly suggests that the calculi were the result of the infective processes.

The case raises many questions of the deepest interest from the point of view of immunity and bacteriology generally. That typhoid bacilli have been passing probably almost continuously for this long period from his gall bladder through the greater part of the alimentary canal, yet without causing recrudescence of the disease, and without producing any marked deleterious influence on the health, is itself a sufficiently striking fact.

In the majority of such cases which have been examined, there is evidence that by a process of absorption of the bacilli or their products immune substances are formed, and are thrown into the blood, the serum acquiring marked agglutinating properties.

In this case, however, the serum does not possess more agglutinins than are frequently present in the blood of healthy individuals, which suggests the possibility that here the absence of bad effects may be due to a local immunity. The fact that serum possesses such low agglutinating properties that it would have been reported as a "negative Widal" by most laboratories is an important point to be noted in the investigation of such cases, for it shows that a negative reaction does not exclude the possibility of a case being a typhoid carrier.

Another point of great interest is the extraordinary change which may take place in the bacterial flora of the faeces. A comparison of the results of plating the faeces in this case with those obtained under parallel conditions from normal cases, shows that there is an enormous reduction in the total number of organisms present in the faeces, and that the normal inhabitants have been altogether, or largely, replaced by the typhoid bacillus. Of the mechanism by which this is brought about we have no knowledge. It is hoped that the further investigation of this and similar cases may yield interesting results.

REFERENCE.

¹ A. and J. C. G. Ledingham, Typhoid Carriers, BRITISH MEDICAL JOURNAL, January 4th, 1908.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL.

SUGAR HEADACHES.

I HAVE been struck for a long time by the number of so-called "bilious attacks" which are due to taking sugar in excess. The sugar standard of Nature is the quantity in milk—that is, a baby or young animal can thrive on the amount found in the "perfect food." But the sweetness of milk is exceeded greatly in our tea and coffee, to say nothing of the starches (converted into sugar in the adult) not found in milk. Again, the quantity of sugar in an average body is said to be two and a half teaspoonfuls, that of salt being about one. Think of the pinch of salt we take to maintain the average, and the enormous amount we take of sugar and starch during the day! Many people take five to seven cups of tea and coffee, each containing two to three heaped teaspoonfuls of sugar, and sometimes condensed milk (also sugary) as well. This excess is converted into lactic acid, which irritates the terminations of the gastric vagus and sets up the well-known but badly-named "bilious attack," with its distressing symptoms of "megrim," of which the commonest is hemi-headache, generally referred to the left eyeball and relieved by pressure, together with vomiting, giddiness, and depression.

A patient has just left my house who confessed to taking five cups of tea daily with three heaped teaspoonfuls of sugar in each—that is, fifteen teaspoonfuls daily. In addition to this, there is often a large quantity of jam or marmalade daily, with sweets and chocolate at