abeyance, and there is no natural means of getting rid of what is killing the patient—the retention in the large bowel of its decomposing contents. The best, I had almost said the only, chance of saving the patient is to open his cæcum and give exit to the poison which is killing him.

It is as a plea for surgical aid to such cases that this paper is written.

On looking back on the past I can recall several fatal cases in which I think the patient might have had another chance given to him by the aid of the surgeon. For what is the condition of such cases and what the cause of danger? It is no direct action of the bacillus, no essential outcome of the action of that poison which is killing the patient. It is late in the attack: he is completing his third week or has entered on the fourth; the sloughs have already separated or are coming away, and sloughs and fetid discharges are passing from the small into the large gut; in most cases they pass on, causing more or less diarrhoea, which, though it may try the patient, has the undoubted advantage of carrying away the sloughing and putrid discharges; but in some cases they do not pass freely away—they lodge in the large bowel, especially in the cæcum; there they undergo fermentation and decomposition, giving off gases which cause more and more distension of the large bowel as they continue to be given off in increasing quantity. The results of this retention in the bowel of these fetid and decomposing contents are twofold: (1) poisoning of the system by the absorption of septic material; and (2) great and embarrassing distension of the large bowel; either result may cause death; the combined action of both is almost essentially fatal. The only way to save the patient is to get rid of what is killing him—the retention in the cæcum and large bowel of its putrid contents. The only effectual way to do this is to open the cæcum. By so doing we should at once evacuate the colon of its gaseous and putrid contents, relieve the diaphragm, and arrest the process of septic poisoning. It is a bold step, but the condition is desperate, and boldness is called for. Since the idea of thus treating these cases presented itself to me I have had no opportunity to put it in practice. Did occasion arise I should not hesitate to recommend it. Without such relief the patient must almost certainly die, with it he might be saved. I make the suggestion in the hope that some of our hospital physicians who see most of such cases may share my views, and give to these desperate cases the chance which opening the cæcum would offer.

The operation would not be much; not nearly so great as that for perforation; the opening would be a small one; and no more than local anaesthesia would be called for. The result would be great, for the poisonous contents of the cæcum would find direct and immediate exit, and the danger of septic poisoning would be greatly diminished. The large bowel could be washed out, and discharges coming from the ileum got rid of as soon as they came down; the distended colon would return to its normal size, and the main obstacles to recovery be thus removed.

Here, as in the case of perforation, I should urge no unnecessary delay. Do not wait until the patient is *in extremis*, but so soon as there is evidence of interference with the action of the diaphragm, and the long tube fails to give relief, get a surgeon to open the cæcum.

As in the case of perforation, the risk of leaving things alone is enormous, the risk attendant on the operation almost *nil*. Let the patient have the benefit of the doubt and the safeguard of the operation. The chances against the patient if nothing is done are enormous. I am probably within the mark in saying that 90 or even 95 per cent. of such cases die. That is a mortality which justifies us in doing anything that gives a chance of recovery. And, after all, opening the cæcum is a less formidable procedure than opening the peritoneal cavity, which we do not hesitate to do in some cases of perforation.

The important point to be borne in mind is that what we have to deal with in these cases is not so much typhoid fever as septic poisoning; that it is not the ulcerated small intestine with which the surgeon has to deal, but over-distension of the large intestine consequent on the retention in it of sloughs and putrid discharges, which have passed from the ileum into the cæcum. In perforation the surgeon has to operate on a

seriously damaged and diseased small intestine; in these septic cases he would be dealing only with a distended but undamaged cæcum.

REFERENCE.

¹ *Lancet*, February 9th, 1901.

A CASE OF TYPHOID ARTHRITIS PROPER.

By JOHN J. BUCHAN, M.B., CH.B.,

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CASES of enteric fever in which joint complications arise are very infrequent, and I have therefore thought it well to record the following case.

T. M., male, aged 11, was admitted into Middle Ward Hospital, Motherwell, on February 5th, 1900, notified to be suffering from enteric fever.

History.—Except for slight infantile troubles, patient had always enjoyed good health. His present illness began on January 20th with headache and a feeling of cold and weakness. He complained of indefinite pains over the body, but these disappeared in a few days. He was restless and sleepless at nights. His tongue became heavily coated, but he had not suffered from diarrhoea. On January 27th he complained of pain in his right elbow-joint, this was increased on pressure and movement. By February 4th the pain in this joint had passed off, but on February 5th the left elbow-joint became painful.

Condition on Admission.—On February 5th, the sixteenth day of illness, the temperature was 100° F. at 1 P.M., the patient was dull and sleepy, the pupils were dilated, and the conjunctivæ pearly and injected. The abdomen was somewhat distended, but there was neither tenderness nor gurgling on pressure over it. Four typical rose-red spots were seen. The spleen was not palpable, but on percussion it came forward to the anterior axillary line. The tongue was fairly moist and covered with a thick white coat. The heart was normal on examination, but in the lungs occasional sonorous and sibilant rhonchi were heard. The urine contained no albumen, but presented the other ordinary febrile characters. Vidal's test gave a positive reaction on the eighteenth day of the disease.

Progress.—While on admission the patient complained of pain, increased on pressure and movement, in the left elbow-joint, no distinct areas of tenderness over the joint could be made out, and, in particular, pressure over the ends of the bones did not reveal any tender spots. The pain complained of was not at all severe, and no swelling was noticed, but on February 9th, the pain continuing, the joint became distinctly swollen. At this time the forearm was pronated and flexed upon the upper arm. The joint was wrapped up in flannel for the first few days, but it was afterwards immobilised. On February 17th the swelling of the joint seemed to be increasing, and it was resolved to aspirate the fluid; one drachm and a-half of clear serous fluid was drawn off, which on bacteriological examination proved sterile. The aspiration was carried out with strict attention to the details of asepsis. The temperature fell to normal, and for a day or two the joint was not complained of. On February 24th, however, pain was again felt and the swelling reappeared. On March 1st the evening temperature rose to 103° F., with, however, no distinct aggravation of the joint symptoms. On March 2nd aspiration was again resorted to, but this time a purulent fluid was drawn off. This fluid was found to contain a large number of streptococci and a lesser number of staphylococci. Careful search was made for the bacillus typhosus and the bacillus coli communis, but neither of these were found.

With the help of Dr. Wyper I freely incised the joint and washed it out in every part with 1 in 80 carbolic lotion. During this process the cartilages were examined and found intact. The wound was dressed daily for the first few days, and healed in a little over a fortnight. By the time the patient was discharged on May 16th, movement had been almost entirely restored.

Very little notice has been taken of the joint complications of enteric fever. The literature of the subject consists almost wholly of isolated cases appearing from time to time in the medical journals. Keen¹ has recently collected 84 such cases, and has divided them into three classes: rheumatic typhoid arthritis, septic typhoid arthritis, and typhoid arthritis proper. To these classes Dreschfeld² has added a fourth, osteoarthritides, where typhoid osteitis is situated near to and extending into a joint.

Taking these four classes into consideration, the diagnosis in the present case would seem to rest between rheumatic

typhoid arthritis and typhoid arthritis proper, as there were no other pyæmic symptoms present in the case, and there was no evidence of osteitis preceding or accompanying the arthritis. Keen, who introduced it, has used the term rheumatic typhoid arthritis rather vaguely. There is, so far as we know, nothing to prevent a patient suffering from enteric fever suffering from rheumatism also; but considering that we now recognise scarlatinal rheumatism as having nothing to do with true rheumatism, we should be very careful in calling cases which present a passing resemblance to rheumatism, "rheumatic typhoid arthritis." This is well seen by the fact that Keen and Dreschfeld in referring to Freyhaus's³ case consider it to be one of rheumatic typhoid arthritis and typhoid arthritis proper, respectively, and I should be slow to call this case one of rheumatic typhoid arthritis unless there were more than mere pain and swelling of the joint to justify it. Against the rheumatic nature of this joint affection there are the following facts: the boy had not suffered from any manifestation of rheumatism previously and none of his near relations had so suffered; the joint affection was almost wholly confined to one joint; none of the commoner complications of rheumatism, such as endocarditis, were present, and lastly the affection became suppurative. It might be argued that the affection became suppurative on account of the previous aspiration, but the fluid in the joint did not become purulent until almost a fortnight after the first aspiration, and every precaution was taken in aspirating.

The diagnosis of typhoid arthritis proper thus presents itself, and despite the later bacteriological examination this diagnosis must be held. The joint which is most frequently attacked in typhoid arthritis is the hip, and after this the other joints of the lower limb. In Keen's 84 cases the lower extremity was affected in 70, the upper in 17, and both extremities in 7 cases. The most frequent result of typhoid arthritis is spontaneous dislocation owing to the gradual lengthening of the ligaments through the distension of the joint. It was to prevent this result that the joint was at first aspirated. Suppurative arthritis is a rare result.

The bacteriological examinations in this case are of some interest. All our present information and ideas of enteric fever would point to the bacillus typhosus as being the cause of typhoid arthritis proper. It is quite in accordance, however, with the published records of bacteriological examinations to find that in this case at one time a negative result was got, while at another pyogenic organisms alone were found. Too few of these cases have been examined however, to enable us to arrive at any result.

REFERENCES.

¹ Keen, *Surgical Complications and Sequelæ of Typhoid Fever*. ² Dreschfeld, article, Enteric Fever, in Allbutt's *System of Medicine*, vol. i. ³ Freyhaus, *Berliner Klinik.*, 1891, Heft xlii, p. 1.

ON ANÆMIA IN TYPHOID FEVER.

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THE blood in typhoid fever has been studied by many observers and some of its interesting characteristics are now regarded as definite features of the disease. Thus it is well known that the anæmia of typhoid fever differs from the majority of secondary anæmias in the fact that while it presents in uncomplicated cases no evidence of leucocytosis there is generally a considerable increase in the proportion of large mononuclear leucocytes.¹

It is also agreed amongst bacteriologists that in typhoid fever there occurs a great increase in the bacterial fermentation of the intestinal contents. In the anæmia which occurs in a fair proportion of cases of typhoid fever we have, therefore, an opportunity of investigating the relation between the bacterial fermentation with absorption of toxins, and the modifications which occur in the blood of these cases. The bearing of this investigation on the various well-known theories of the primary anæmias need not be enlarged upon here.²

The following case of typhoid fever illustrates these peculiarities in a striking manner, and a consideration of the

observations made in this case seems to throw light on some of the interesting features of the disease.

History.—S. T., aged 30, a cattle dealer, was admitted to the fever wards of the Royal Victoria Hospital, Belfast, under the care of Professor Lindsay. He had a history of previous good health, and was described as a "full-blooded man." His only illnesses were an attack of acute pneumonia and some rheumatic attacks. Before admission he had some diarrhoea and epistaxis, and had been ill for about ten days. The fever ran a rather protracted course, and he was altogether 87 days in hospital. A few of the more important details copied from the clinical notes will be sufficient for our present purpose. On admission (Nov. 26th, 1900) having been ill about ten days, his pulse was 98, and his temperature 102°. He had slight cough, diarrhoea, with fullness of the abdomen, but no pain or gurgling in the right iliac fossa. There were a few spots, and the tongue was furred. The heart sounds were normal; there was some bronchitis; the spleen was enlarged, and the patient was somewhat incoherent in his answers to questions. On November 29th he had epistaxis. On December 8th there was slight hæmorrhage from the bowels. On December 12th there was hæmorrhage from the bowels fairly large in amount. On December 23rd phlebitis was discovered. The temperature for a few days before this had been about 101° at night. It now gradually rose to 104° (this rise was no doubt due to the phlebitis). On January 1st, 1901, the temperature was normal, and remained so for four or five days, and then began to rise again. On January 20th abscesses were discovered about the rectum, and incised. The temperature previous to this treatment was 103°. On January 27th the patient had a rigor; after this the temperature remained normal for some time, with very slight rises. He was noted to have an anæmic appearance. On February 16th small abscesses were found in the axilla, and opened. On February 20th the patient left the hospital. The temperature was subnormal. He was still somewhat anæmic looking, but otherwise was much improved.

This patient had thus a rather protracted attack of typhoid fever complicated by some epistaxis and hæmorrhage from the bowels, and prolonged by a phlebitis and abscesses about the anus and in the axilla. The patient during the illness became considerably anæmic.

For the sake of clearness, the record of this case may be divided into six periods:

No. I.—Early Stage.

November 27th, 1900. Ill about twelve days. Red cells, 4,930,000; white cells, 5,300; hæmoglobin, 88 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	72.4
Small lymphocytes	20.0
Large mononuclear leucocytes	6.2
Transitional leucocytes	1.4

This was practically a normal blood, except that the white cells were somewhat diminished in number, and the large mononuclear leucocytes slightly increased.

No. II.—Anæmia Commencing; No Inflammatory Complications.

December 6th, 1900. Red cells, 3,000,000; white cells, 4,200; hæmoglobin, 67 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	51.2
Small lymphocytes	37.6
Large mononuclear leucocytes	8.4
Transitional leucocytes	2.8

The hæmoglobin and red blood corpuscles were now considerably diminished. In the differential count the lymphocytes and mononuclear leucocytes were increased in percentage.

No. III.—Anæmia persisting with Phlebitis.

December 20th, 1900. Red cells, 2,900,000; white cells, 4,600; hæmoglobin, 65 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	66.0
Small lymphocytes	30.6
Large mononuclear leucocytes	3.4
Transitional leucocytes	0.2

Although the hæmoglobin was somewhat lower than in former observations, the leucocytes had risen somewhat, while the percentage of polymorphonuclear leucocytes had considerably increased. I was at first at a loss to understand the meaning of this alteration in the blood picture, as it was the reverse of what had been expected until Dr. Mandale Byers, the house-physician, on investigating this anomaly, discovered that the patient had developed a phlebitis of the left femoral vein. This explained also the rise of temperature which occurred at this time. It is, however, to be noted here that the number of leucocytes per c.mm., although greater than in the former count, was still much less than is usually found in an inflammatory condition. The increase, however, took the form which is observed in ordinary inflammation.

No. IV.—Anæmia Increasing; Phlebitis Disappearing.

January 2nd, 1901. Red cells, 2,800,000; white cells, 3,500; hæmoglobin, 60 per cent.

Differential count:

	Per cent.
Polymorphonuclear leucocytes	51.0
Small lymphocytes	40.0
Large mononuclear leucocytes	8.5
Eosinophile leucocytes	0.5

Here the hæmoglobin is still lower, while the white cells have somewhat decreased again. The percentage of polymorphonuclear leucocytes