DYSENTERY AT GALLIPOLI.

Sir,—The letters from Captains Bahr and Bartlett, R.A.M.C., on dysentery at Gallipoli, which appeared in your issue of September 22nd, were of considerable interest to me.

I had an opportunity of studying the epidemic in question from the clinical aspect at Mudros and Suvla Bay during July and August, 1915, and later from the bacteriological aspect at the Central Laboratory, Alexandria.

My conclusions were embodied in a paper which appeared in October, 1916. I am in complete agreement with Capt. Bahr that the large majority of the cases were of amoebic origin. This belief was supported by the clinical features characterizing the cases with which I came in contact, the response to emetine treatment, the findings during microscopical and cultural examination of the stools, the results of serological tests, and the appearance presented by the intestines of fatal cases.

Clinical Features.—All cases occurring amongst the men under my charge were characterized by the insidious apparatus and, as was supposed to be characteristic of dysentery. Symptoms of severe diarrhoea with abdominal pain heralded the onset, and it was not for twenty-four to forty-eight hours or later that the patient, frightened by the appearance of blood in his motions or compelled by the increasing severity of the symptoms, reported symptomatically.

Response to Emetine.—During the early stages of the epidemic the patients rarely received emetine until their arrival at the base, and during this period the cases admitted to hospital in Egypt were very severe, showing as usual abundant blood and mucus in the stools. Later, when emetine was injected at the field ambulances and stationary hospitals, the type of case coming under our observation at the base was certainly much less severe. Many showed no blood in the stools and some were completely free.

Examination of Stools and Serological Tests.—In my series of cases 12 per cent. were shown to be pure amoebic infections, in 7.8 per cent. dysentery bacilli were isolated, and in 2.6 per cent. both E. histolytica and dysentery bacilli were found. Therefore, of the positive cases, the amoebic far outnumbered the bacillary.

How can the failure to detect the causative agent in such a large number (over 70 per cent.) of cases be explained? It is due to incomplete examination or to the disappearance of the latter previous to the time of examination? The greater number of negative cases in this series were examined on several occasions, so that I do not believe that any considerable error was introduced owing to incomplete examination. The conclusion reached was that the majority of these cases were entamoebic in origin, the entamoebae having disappeared from the stools as the result of emetine treatment.

This view was supported by the fact that serological tests carried out during December, 1915, and January, 1916—months during which amoebic dysentery was presumably reaching its ebb—resulted in only 30 per cent. being shown to be of bacillary nature. Therefore even at this period of the year the amoebic cases outnumbered the bacillary.

Appearance of Intestines.—Evidence of the amoebic nature of the lesions is afforded by the appearance of isolated nodular projection and flask-shaped ulcers separated by normally mucous membrane. This appearance was only presented by the intestine in the early stages of the disease as secondary bacterial invasion of the ulcers soon alters the appearances, and gives rise to the picture of a more or less diffuse inflammation, with numerous irregularly shaped ulcers. In an advanced case of the disease, therefore, the amoebic origin of the lesions can only be proved by microscopical demonstration of the parasite in the intestinal wall.

In this regard I may mention that the amoebae-like appearances presented by certain phagocytic and vacuolated cells, alluded to by Captain Bahr, was well recognized, and reference was made to them in the paper previously mentioned.¹

I was privileged to attend a large number of post-mortem examinations carried out by Captain Bartlett on fatal cases of dysentery, and of these the large majority showed, either to the naked eye or microscopically, typical amoebic


RUPTURE OF UTERUS.

Sir,—Dr. H. Davies-Jones’s concluding sentence in his letter on the above subject in your issue of September 22nd should be sufficient to warm off most of us; but I mean, with your permission, in all diffidence, to enter the arena.

First, I desire to add my congratulations to Dr. Rigden on the eventual recovery of his patient, though childless and without her uterus. The history and Dr. R’s statement show us that it was a case of flat pelvis, though to what extent of capacity we are not informed, nor, what is equally important, do we know the disproportion between child and pelvis, which cannot be estimated without some data as to weight of child as well. What a text for the advocates of antenatal treatment this case affords! and, in passing, one may note that according to the new rules of the Central Midwives Board, 1916, the midwife erred in not advising her patient to have medical advice before labour.

The important decision as to immediate turning, or, alternatively, the application of forceps in flat pelvis, depends largely on an unfavourable or favourable position of the head respectively, but, personally, the forceps in nearly all cases is my choice. Probably the most salient point in this remarkable case for comment and our guidance to treatment is the fact that there was no pulsation at the umbilical cord, subsequently, the child being dead, immediate perforation, and embrodyome if necessary for easy extraction, was indicated.

I do not think it fair for any one not present at the case to comment on the subsequent surgical treatment, but I think it is arguable that the immediate removal of the placenta per vaginam might have saved the abdominal section, bearing in mind in this connexion that figures of mortality after uterine rupture by themselves are insufficient to decide the relative claims of plugging and traction as against immediate abdominal section. I am, etc.,

Norwich, Sept. 8th. ______.

ARTHUR CROOK.

RECURRENT OF ADENOIDS AND TONSILS.

Sir,—It is one’s common experience to examine children with respiratory deficiency and to refer them to the first instance to a deflected septum, with the usual turbinate deformity. Very frequently there is a history of operation for removal of tonsils and adenoids, with, naturally, poor results, as judged by the unaltered mouth breathing and unchecked facial deformity. Heredity plays a very prominent part, with the result that “asthma in the family” may often be interpreted as inherited nasal deformity; and unfortunately, so far, very little can be done in children to remedy this abnormality, with its usual resulting train of signs and symptoms.

A few days ago I became possessed of a remarkably well-preserved skull, recently unearthed on the site of the Roman settlement of Caerwent. There is well-marked septal and turbinate deformity, and the usual alteration of the palate, antrum, etc. General skull shape square, angle, and well-formed nasal bones presume a male Roman, I am, etc.,

Newport (Mon.), Oct. 6th. ______.

J. LEWIS THOMAS.

TREATMENT OF ACUTE GONORRHOEA.

Sir,—The article by Captain Lamb, R.A.M.C., in the British Medical Journal, October 6th, 1917, calls for some comment, since the electrical process is described as similar to my method. I must point out that he made deviations from my method, and these doubtless affected his results. First, Captain Lamb has employed a silver catheter, and he must be aware that during electrolysis this instrument is being dissolved, and silver ions are being driven into the lining of the urethra. He has, therefore, been treating these cases by silver ionization—a process I have never described or recommended. The second important variation is an arrangement of an

¹ Quarterly Journal of Medicine, April, 1917.
² British Medical Journal, September 8th, 1917, p. 299.