by the mouth in keratin capsules, but this also has failed. We have found most promise of success in the internal administration of small doses of calomel over a prolonged period associated with a mild morning saline.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, May 20th, 1916. ² The Lancet, June 10th, 1916.

A CASE OF AMOEBIIC ABSCESS OF THE LIVER OCCURRING TWENTY YEARS AFTER THE ORIGINAL ATTACK OF DYSENTERY.

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CLINICIANS have often noticed that abscess of the liver may follow many years after an attack of dysentery, or even appear with no history of dysentery at all. Such occurrences have, up to the present, been very puzzling, but recent work carried out on cyst carriers helps largely to explain them.

It has been shown that a person may become a contact carrier and present *Entamoeba histolytica* cysts in his stools without ever having had dysentery or even diarrhoea, and, further, that a person who has had dysentery may become a relapsing carrier and go on discharging cysts regularly or irregularly for many years. Either of this class of case may develop an abscess in the liver at any time, and there is no reason why they should not, as the presence of cysts in the stools indicates that they are still the subjects of amoebiasis. What is still difficult, however, to explain is why some cases develop abscesses and others do not. There would even seem to be a geographical predilection, because in India and other places, for example, they are common sequelae of amoebic dysentery, while in other parts of the world, though there is plenty of dysentery, they are distinctly rare. Further, why should a man the subject of amoebiasis live for years and then suddenly get liver symptoms and develop an abscess? These are points that still require solution.

Recently I have had the opportunity of fully studying a very interesting case of amoebic liver abscess appearing years after a dysenteric attack, and as it raises many points of interest no apology is necessary for publishing it.

A ship's chief engineer, aged 54, was admitted into the Seamen's Hospital, London School of Tropical Medicine, under my care, complaining of pain over the liver and stomach, with vomiting and hiccough.

History.

He was born in England. He went to sea at the age of 22, and has followed the sea ever since with the exception of three years, when he lived ashore in England. He has travelled all over the world—to India, Australia, South America, but not to Africa. In his time he had drunk a fair amount of alcohol, but lately had been more temperate. He had had gonorrhoea in his youth, and, at the age of 25, a chancre, for which he was never treated; apparently he had no secondaries. He had had malaria several times, slight attacks. He had dysentery twenty years ago at Port Said, with typical clinical symptoms—blood and mucus, tenesmus, straining, etc. He was treated in hospital there, and got better in a week or two. After that he had slight touches of diarrhoea for two or three years; thereafter all signs of bowel irritation disappeared. He is perfectly certain that he never had dysentery again—that is, a fresh infection. In September, 1915, he passed blood. This was found to be due to piles; he was operated on and cured.

Present Illness.

Nine weeks previous to admission to hospital he began to get feverish attacks, with general aching all over the body, diagnosed as influenza and treated as such. He had also slight vomiting. A week later he developed ague attacks, shivering followed by sweating, with headaches; this lasted four weeks. He was treated by his own doctor with quinine. A week later hiccough developed, with pain in the epigastrium and under the right costal margin. As his condition did not improve, and as increasing weakness and loss of weight also became apparent, he was sent into hospital, arriving there on July 5th, 1916.

State on Examination.

The patient's mouth was in a very septic condition, an extreme degree of pyorrhoea alveolaris existing. The tongue

was dry and coated, and there were numerous small ulcers in the mouth. The temperature was 101°. There was nothing abnormal in the lungs, and no dullness at the right base. The heart was a little enlarged, the sounds were very faint (? fatty). The pulse was irregular, missing beats regularly, rate 90 to 100. Abdomen: Pain on pressing epigastrium; liver enlarged, upper border fifth and sixth ribs, lower border three inches below costal margin; spleen 9 to 11, not enlarged. No tenderness over colon, nothing in groins. Urine: Specific gravity 1020, no albumin, blood, or sugar. Faeces: No amoebae, cysts, or flagellates; no ova.

Blood examination:

Red cells	...	...	...	...	4,800,000
White cells	...	...	...	...	14,000
Haemoglobin	...	...	...	...	100 per cent.
Differential count:					
Polymorphonuclears	...	...	...	...	60 per cent.
Large mononuclears	...	...	...	...	11 ..
Lymphocytes	...	...	...	...	27 ..
Eosinophile	...	...	...	...	1 ..
Mast cells	...	...	...	...	1 ..

No malarial parasites or pigmented leucocytes.

Diagnosis.

Hepatitis and gastritis, probably septic in nature.

Treatment.

All his teeth were removed at once; Mr. Vaughan, who did this, agreeing that the trouble might all be coming from the mouth condition.

Progress.

July 6th. A little better. July 10th. Temperature keeping up, two or three small rigors. Feels better, however. Faeces: No cysts or amoebae. July 13th. Much better; no more rigors, no sweating; temperature down a little. Leucocytes 14,000. Faeces: Again negative.

July 17th. Worse, rigors again increasing in severity; pain over liver on shaking him. Tender on deep pressure over lower axilla (? a liver abscess starting). Leucocytes, 15,000; no malarial parasites. Faeces: Again negative. Give him emetine, beginning to-night, one grain; mouth quite clean now. There does not seem to be any source of infection there.

July 18th. A dead amoeba and two *E. histolytica* cysts found in faeces to-day. This clears up the diagnosis and verifies the suspicion of an abscess forming in the liver. No rigor to-day.

July 20th. Temperature still raised; no further rigors, however, and no more sweating. The patient feels more comfortable, and says he has lost the liver pain. Sick in the afternoon. Decided that surgeon should see him and explore his liver for an abscess the following morning. No further sickness after afternoon, but pulse became very irregular in evening.

July 21st. Seized with a sudden heart attack at 2 a.m. and died at 2.45. Owing to the irregularity of his heart, and also because of its weakness, the patient had been on digitalis since July 14th.

Table giving Details of the Emetine Treatment and Results of the Examinations of the Faeces and the Leucocyte Counts.

Date.	Dose and Drug Employed.	<i>E. histolytica</i> Free.	<i>E. histolytica</i> Cysts.	Leucocytes.
1916. July 5	..	—	—	14,000
" 10	..	—	—	
" 13	..	—	—	14,000
" 17	Emetine hydrochloride subcutaneously	—	—	15,000
" 18	Ditto ..	+	+	
" 19	Ditto ..	—	—	
" 20	Ditto ..	—	—	16,500

The temperature is shown in the chart.

Autopsy.

The patient died at 2.45 a.m., July 21st, 1916. Post-mortem examination 4 p.m. same day.

External Appearances.—Body well nourished; good layer of fat; old scars on legs look like specific; no scars on penis; otherwise nothing to note.

Head, neck, and spine not examined.

Thorax.—Pleurae: Right, no fluid, adhesions between lung at apex; left, no fluid, no adhesions. Right lung, 1 lb. 9 oz., adhesions at apex; no signs of tubercle, however; body of lung congested, and lower lobe oedematous; no pneumonia. Left lung, 1 lb. 6 oz., similar to right.

Heart.—Pericardium: No fluid, adherent to wall of heart by old fibrous adhesions. In addition to this, a recent exacerbation of acute pericarditis with fresh lymph. Muscle substance very soft and flabby, advanced fatty degeneration. Valves: Nothing abnormal on right side. On left, aortic nil; mitral, old sclerosing endocarditis on both cusps. Aorta: Early atheroma.

R = Rigor. A = Intramuscular injection of emetine, gr. j. D = Died.

Abdomen.—No fluid, no peritonitis.

Liver.—Large, edge lying 3 inches below the costal margin. In a condition of early polylobular cirrhosis. Adhesions between it and the diaphragm rendering its removal difficult. After its removal from body a fairly large abscess became apparent, this being about the size of an orange, and measuring roughly 4 by 5 inches. Its situation was at the junction of the upper and right margins—that is, somewhat to the right of the dome of the right lobe. Its external wall was comparatively thin, and it was here that the chief adhesions between liver and diaphragm had existed. On making an incision into the abscess, chocolate reddish coloured pus, typical of amoebic liver abscess pus, escaped. A microscopic examination of a scraping of the wall made at once showed living and active *Entamoeba histolytica* in the pus. The general substance of the organ exhibited cirrhotic and fatty changes. Weight of liver, 7 lb. 2 oz.

Spleen.—14 oz., enlarged, and very soft. Spleen of sepsis, not definitely pigmented.

Kidneys.—Right 9 oz., left 9½ oz. The capsules stripped easily in both. Substance of organ cloudy swelling.

Intestines.—Small: No ulceration or any lesions. Large: No thickening of wall noticeable. Lumen practically normal, with the exception of the following: In the caecum, just at its junction with the ascending colon, there were too little pockets or crypts, with their mouths about half the circumference of a threepenny-bit. Above this there were some very thin places, which looked like healed scars of ulcers. Some of these were also present in the transverse colon, none in the descending or rectum. Over a considerable part of the mucous membrane some greyish-black spots the size of a fair-sized pin-head were seen. These had a glazed appearance.

In the rectum were four small haemorrhagic spots, something like the early lesions of bilharzia disease. These were very small, only a little larger than a millet seed. The mucous membrane over them did not appear to be ulcerated. Scrapings revealed no signs of bilharzia eggs, nor any active amoebae. They were situated within two inches of the anus.

Stomach.—Mucous membrane congested; chronic gastric catarrh present.

Bladder.—Nothing to note.

As far as one could judge from the appearance of the abscess, the character of the pus, and the presence of the living and active amoebae, the age of the abscess seemed to correspond to the onset of the clinical symptoms some nine weeks before. There were certainly no signs of chronic encystment about it. The immediate cause of death was due evidently to the pericardial condition influencing the heart and to the general septic condition, rather than to the abscess, which, after all, was not of an excessive size, and which up to the time of death had not caused any serious complications.

Microscopical Examination.

Sections of the abscess wall showed the presence of amoebae. The deep crypt or pocket in the caecum was lined with mucous membrane, with glands, the intervening spaces being largely filled with a small celled infiltration of uninuclear cells; outside the mucous layer was a fibrous tissue with little or no signs of vessels. Further out again the arterioles seemed to have very thickened walls, very suggestive of syphilis, quite a likely possibility from the clinical history. No amoebae were found in this pocket.

The other pocket was much shallower, more a depression than a real pocket; it was also clothed with a layer of mucous membrane; no amoebae were found in it also. In between the two crypts was a large mass of muscle, its position there being somewhat difficult of explanation. It looks as if there had been extensive ulceration, and that this piece of muscle had been cut off and isolated in the process of healing.

The sections of the thinned-out pieces of bowel showed a

faulty mucous membrane with great thinness of the wall. Possibly they represent scars of previous ulcers.

For these microscopical sections I am greatly indebted to Dr. A. C. Stevenson of the Wellcome Institute who cut and prepared them, and also for his help in discussing their significance.

Several very interesting facts are brought out in this case. The practical absence of any gross lesions in the large intestine would seem to point to the patient's statement, that he had only had one attack of dysentery, and that twenty years ago, being correct. Though this is so, the presence of an amoeba and two *E. histolytica* cysts in the faeces on one occasion meant that he still had a bowel infection with the specific parasite, and the fact that he had an abscess in the liver full of *E. histolytica* also supports this. Where in the bowel the parasites were one cannot say. There were certainly no signs of them in the pockets or crypts. They may probably have been present in the small haemorrhagic spots in the rectum. These unfortunately could not be certainly identified after the material had been hardened, and to have found them by sectioning would have involved serial sections of some inches of bowel—a too laborious proceeding. It is also quite possible that they were present in some minute lesion, not apparent to the naked eye, but, if so, this must have been very minute, as the bowel was carefully examined by a hand lens to see if any such place could be detected.

Another point of some interest is that the amoebae were still alive in the pus of the abscess thirteen hours after death, and incidentally also after 4 grains of emetine hydrochloride had been administered hypodermically. Once an abscess has formed in the liver, as in this case, it is difficult to see how the emetine can reach the amoebae.

Much attention has recently been bestowed, and rightly so, upon the danger of chronic amoebic carriers spreading infection to others. This case is a very striking example of the danger of the carrier to himself, and brings forward once more the necessity of properly treating such cases. Fortunately we now possess in emetine bismuth iodide a drug which sterilizes chronic cyst carriers with a certainty not attained by any previous methods.* Since Dobell and myself published a series of cases treated by this drug¹ further results showing its efficacy have come to hand. The results obtained, for example, at Walton Hospital recently, results which have been annotated and published by Dobell,² are most striking, and similar successes have been obtained at other places.

It is therefore of the utmost importance that all people who have had dysentery, even though it may have occurred years before, should have their stools thoroughly examined on several occasions to make certain that they are not amoebic carriers; if they are found to be so, they should at once be treated in order to obviate the danger of a subsequent abscess in the liver.

REFERENCES.

¹ *Lancet*, August 19th, 1916. ² *BRITISH MEDICAL JOURNAL*, November 4th, 1916, p. 612.

* It is essential, however, that the drug be given as the powder in a gelatin capsule, and not in any compressed form. Since writing this paper I have had a case in which "steartotes" of it were passed in the faeces absolutely unchanged, and the same may take place with keratin-coated tablets.