Interaction between the host and parasite.—Three types of reactions are mentioned in the literature—(1) Immunity reaction : This provides a scientific test for the diagnosis of hydatid disease within certain limits. In this case this test was not done. (2) Leucoblastic reaction : There is as a rule increase of eosinophil leucocytes which is regarded as a strong presumptive evidence of hydatid invasion, but in this case, I am surprised, the count has never showed rise in eosinophils more than the average even on repeated examinations. (3) The toxic reaction : (*a*) Urticaria. (*b*) Local inflammatory processes from direct contact with hydatid fluid (peritonitis). Both (*a*) and (*b*) were absent in this case. (*c*) Chronic toxic effects, *e.g.* wasting and anæmia. These were prominent here. (*d*) Rise in blood pressure. This is not mentioned in the literature and is suggested by me.

Summary

1. A case of primary echinococcic cyst of broad ligament is reported.

2. Anæmia and rise of blood pressure were associated with the cyst.

PNEUMOPERITONEUM IN THE STUDY OF HEPATIC ABSCESS

By H. B. LAL

LIEUTENANT-COLONEL, I.A.M.C.

J. D. S. CAMERON and N. A. LAWLER have given aspiration with air replacement a place of very great importance in the treatment of liver abscess. Introduction of air into the abscess cavity was to assist in the study of the progress made by the latter in the process of healing, by radiological means. Although Cameron during his tenure as a physician consultant to AHQ(I) spared no efforts to popularize aspiration with air replacement as a method of treatment, it is felt that the medical profession has not taken to it with any great enthusiasm, there being certain objections to the procedure recommended by him :—

(*a*) It is not outside the experience of most physicians in the tropics that emetine in combination with organic compounds of arsenic and of iodine would resolve a very large proportion of hepatic abscesses, and that aspiration has to be resorted to only in exceptional cases in whom the abscess has reached a size when spontaneous absorption, after destruction of the amœbæ and the secondary organisms, is not physically possible, or when an abscess originally amœbic has been converted into a pyogenic one by the secondary invader taking on a more prominent rôle.

(*b*) It has been demonstrated by McCallum that amœbæ are found only in the margins of the living and not in the necrotic tissues of the abscess, unless the abscess has been opened to the air. He points out that this is because of their need for oxygen. An amœbic abscess presents very few amœbæ even in the walls in a closed state, but their number greatly increases both in the walls and in the pus after being opened to the air. Air replacement after aspiration of the necrotic material can only give a further stimulus to the amœbæ in the walls to increase in number and also to flourish in the remnant pus, and thus render the task of amœbicidal drug all the more difficult in dealing with them in the necrotic contents of the abscess cavity.

(*c*) Injection of air into a cavity, the volumetric size of which is not known, is not altogether free from risk. Injection of too great a volume of air might by increase in tension force the pus further into the healthy tissues or might cause a tear of the latter. Further, it is quite logical that absorption of air by inflamed abscess walls could not be efficient and the continued presence of air might delay the contraction of the cavity and its ultimate healing.

Cameron in common with most authors has recommended the site of election for aspiration to be the 8th to 10th interspace in the midaxillary line. But it is common experience that, more often than not, the abscess cavity proves to be very evasive, necessitating puncture of the liver in various directions before the pus can be struck, and then it cannot be definitely ascertained if the needle has entered the main abscess cavity or merely a subsidiary one.

To overcome all these difficulties, the author is anxious to introduce his method of the study of hepatic abscess by radiological examinations after induction of pneumoperitoneum. The author further feels that the treatment of every case of hepatic amœbiasis should be controlled by this method, whether the treatment is purely medical or aspiration has become necessary.

After insertion of air into the peritoneal cavity, an antero-posterior skiagram of the lower chest shows up the outline of the liver with great clarity along all its borders. The shadow of the superior margin of the liver becomes distinct from that of the diaphragm, separated by a narrow line of radiotranslucency between the two, with a break where the bare area of the liver is directly attached to the diaphragm. The lateral view shows up the break caused by the bare area even better. In the pathological lesions of the liver, study of the lateral views requires a clear knowledge of the appearances, as the picture is composite of both the halves of the diaphragm, and the variations from the normal have to be read mostly in relation to the shadow of the right half of the diaphragm.

The pathological appearances may take the following forms :—

1. Generalized enlargement of the liver, with a normal contour and a total absence of any adhesions, in cases of early hepatitis, with no definite tendency as yet of abscess formation.

2. Localized prominence of one or the other part of the contour without any adhesions to the diaphragm or abdominal wall in early precipitations of an abscess.

3. Localized prominence with neighbouring adhesions to the diaphragm or the abdominal wall, according to the situation, along with deformity of the diaphragmatic dome, in cases of definitely established hepatic abscess with a tendency to point in one direction or the other.

4. Generalized adhesions all round the liver resulting from a generalized hepatitis of a severe or prolonged type. This appearance is nearly always left with a persistent pain and tenderness in the hepatic area, with a slightly palpable liver, in spite of a most thorough treatment. This sequela can be explained either on the grounds of a partial passive congestion due to fibrotic constriction around the hepatic vein or due to a persistent low degree of hepatitis with cirrhotic tendencies.

5. Localized prominence of the lower surface of the liver in case the abscess is localized towards that surface.

6. Central abscess has to be concluded on clinical grounds, the presence of a more marked enlargement in one lobe, in the absence of any in the other.

It is within experience that the extent of the adhesions and their site are quite indicative of the ultimate prognosis of the case from the point of view of recovery from pain and tenderness in the hepatic area. Further, as the air in the peritoneum remains visible under the *x*-ray for about a week, the progress of the hepatitis or hepatic abscess can be watched in the early stages of the treatment on a screen.

The advantages of this method of serial study of hepatic amœbiasis lie in :—

1. Exact differentiation between a generalized hepatitis and a localized abscess, even at a very early stage of the latter.

2. Study of the progressive changes in the size and shape of the liver before, during and after the treatment, without in any way interfering with the pathological condition itself.

3. Exact localization of the hepatic abscess or localized hepatitis by study of an A.P. and a lateral view, its variations in size under treatment and the determination of the site where aspiration should be done if this becomes necessary.

4. Differentiation of hepatitis and hepatic abscess from other conditions of the liver, *e.g.* echinococcal cysts, new growths, etc. While the former will retrogress under appropriate treatment, no such change will be seen in the latter.

5. Prevention of adhesions to the diaphragm and the abdominal walls, which contribute so much to the remnant pain and tenderness in the hepatic area.

6. Differentiation of the supra-diaphragmatic lesions from the sub-diaphragmatic ones—the best example is when the shadow of a collapsed lower or middle lobe of the right lung causes a continuous shadow with the liver. Pneumoperitoneum brings the diaphragm and the liver into contrast with that of the collapse. The presence of a long-standing inflammatory lesion at the base of the lung, however, produces adhesions on both sides of the diaphragm and may reduce the value of this method of differentiation.

7. Author's experience that pneumoperitoneum accelerates the recovery of the patient and contributes towards his well-being during the course of the treatment.

Certain objections have been raised to the induction of pneumoperitoneum in cases of hepatic abscess. The main fear has been one of tearing of adhesions around an abscess, already pointing either towards the diaphragm or the abdominal wall. Such an event has never materialized in the author's experience. Further, it may be pointed out that the introduction of 250–300 cc. of air into the abdominal cavity rendered the intra-abdominal pressure positive merely to the extent of +4 to +5 mm. of water, and this pressure is never enough to cause any separation of adhesions. A further precaution is taken, of schooling the patient to relax his abdomen, without which even the induction of pneumoperitoneum becomes difficult in the absence of a paralysed diaphragm, as any attempt at straining the abdominal muscles stop the flow of air. The question of infection of the peritoneum does not arise in the face of the safety with which the physicians have used this procedure in the treatment of pulmonary tuberculosis.

Technique

The patient usually requires no premedication. He is asked to evacuate his bladder; the lower abdomen is shaved and washed with soap and water, and cleaned with spirit. The part is then painted with acriflavine in spirit, covered with a sterilized towel till the operation is begun.

The pneumothorax apparatus is set with the water having been displaced to the distal bottle, and the air and manometer circuits completely closed against each other and the delivery tube.

The operation consists of primary induction of local anæsthesia at the midpoint between the pubis and the umbilicus. It must be stressed that the anæsthetic fluid must be injected with a small bore hypodermic needle and properly

infused under the skin into the underlying integuments and the peritoneum. After the local anæsthesia has taken effect, the pneumothorax needle is boldly inserted through the original puncture, till the peritoneal coat is pierced, when all resistance seems to give way. There is hardly any danger of puncturing or damaging the gut wall. The stylet is withdrawn and the needle connected to the delivery tube.

At this stage the patient is asked to relax the abdominal muscles completely. This is very important because any straining on the part of the patient will effect the manometric readings as well as the flow of air when the pneumoperitoneum is being induced.

Now the manometric circuit is opened to the delivery tube, and if the abdomen is well relaxed, a negative pressure of —2 to —4 will be registered, with slight variations with respiration. The manometric circuit is now completely closed and the air circuit opened to the delivery tube. The air will start flowing into the peritoneal cavity and can be heard hissing with the aid of a stethoscope and appearance of tympanic resonance in the abdomen. When 250 to 300 cc. have been injected, the circuit is closed and the needle withdrawn, the puncture wound being sealed with collodion.

The patient is then asked to sit upright for about ten minutes to enable the air to rise up below the diaphragm and around the liver and stomach. It is important that screen examination is done before the *x*-ray pictures are taken to determine the best views for the same.

The procedure could be repeated when re-examination is required.

For details of *x*-ray appearances *see* figures 1, 2 and 3 in plates XVI to XIX.

Summary

The idea of diagnosis, estimation and observation of the progress of amœbic hepatitis by serial skiagrams after induction of pneumoperitoneum is presented.

GENERALIZED CYSTICERCOSIS CELLULOSÆ

By C. J. DAVE

From the Pathology School, Grant Medical College, Bombay

CYSTICERCOSIS CELLULOSÆ is the term given to the somatic infestation of man by *Cysticercus cellulosæ,* the larval or bladder worm stage of tapeworm *Tænia solium.* Usually man is the definite host for the adult worm, being infested by eating measly pork; whereas in this condition he becomes the intermediate host for the embryos, the infestation occurring *via* food and drink contaminated from outside or from lack of personal hygiene, or by reverse peristalsis, which helps in liberating the embryos in the upper part of the intestinal canal.

Cysticercosis cellulosæ cannot be considered a unique condition in man. It has been known since 1558 and several hundreds of cases were on record from Central Europe. During the first half of the nineteenth century it was seen in 2 per cent of autopsies in Berlin. This was at a time when uncooked ham was very popular in Germany. With improved hygienic conditions it became a rarity, so much so that a practitioner tended to forget it. Recent interest was revived by MacArthur (1934) who reported 22 cases of epilepsy in apparently healthy soldiers returning from India. Ten cases were due to cysticercosis. In another series of 82 cases of soldiers returning from India, 22 were proved to be due to cysticercosis. A number of cases was then reported by many other workers. The most notable reports are by Dixon and Hargreaves (1944) who have followed 284 cases of cysticercosis for ten years. Cases have been recorded from Mexico (Mazzotti, 1944), China (Ch'in, 1933; Chung and Lee, 1935), and other countries. In India there are very few recorded cases. The first record was an autopsy report by Armstrong (1888). From 1888 to 1947, there are about 12 reports, 7 being clinical cases and 5 autopsies, one case only being followed both clinically and by autopsy examination. The first clinical report was by Williams (1906), a case of cysticercosis of the tongue. Second was by Elliot and Ingram in 1911, a case of sub-conjunctival cysticercosis and another similar case by Wright in 1923. The fourth case was recorded in 1935 by Dogra and Ahern, a case of cysticercosis in a British soldier residing in India. Fifth and sixth cases were of somatic tæniasis reported by McRobert in 1944 and Subramaniam in 1946. The seventh and the last known recorded case was a case of cysticercosis resembling myopathy by McGill in 1947. The first autopsy record is by Armstrong in 1888, second by Williams in 1906, third by Tirumurti in 1911, fourth by Krishnaswami (1912) and fifth by Campbell and Thomson in 1912, the last being the case that was followed clinically and by autopsy examination.

Most of the above recorded cases in India are from Madras and South India, while those reported in Dixon and Hargreaves' (1944) series were from the Punjab and the United Provinces. Cases of cysticercosis are rare in Bombay. It may be added here that Dr. P. V. Gharpure, Professor of Pathology of this institution, came across a case of cysticercosis in 1947; the case had been referred to by the Tata Memorial Hospital, Bombay. Recently an autopsy case of generalized cysticercosis cellulosæ was encountered in Bombay again, which supplies the first autopsy record of its kind here. A brief report of this case is given below :

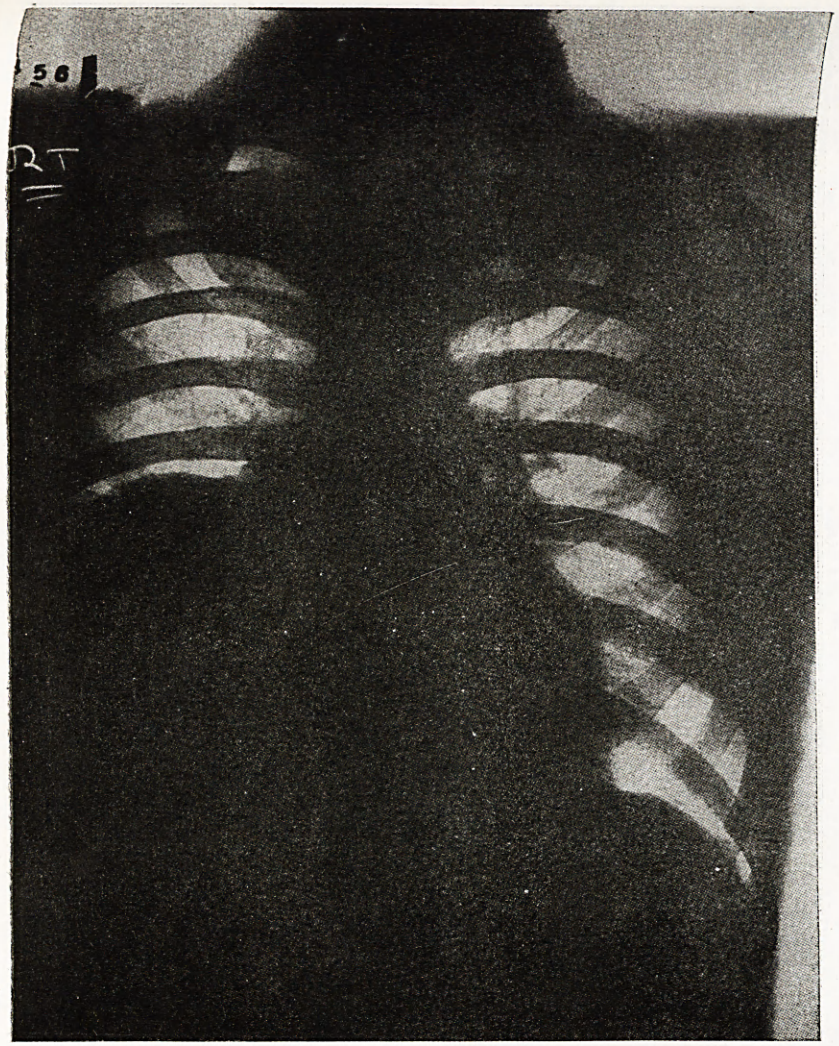


Fig. 1.—Large hepatic abscess—with adhesions to lateral chest wall.
(*a*) Normal A.P. view.

Fig. 1.—(*b*) A.P. view after pneumoperitoneum.

PLATE XVII

PNEUMOPERITONEUM IN THE STUDY OF HEPATIC ABSCESS : H. B. LAL. (O. A.) PAGE 90

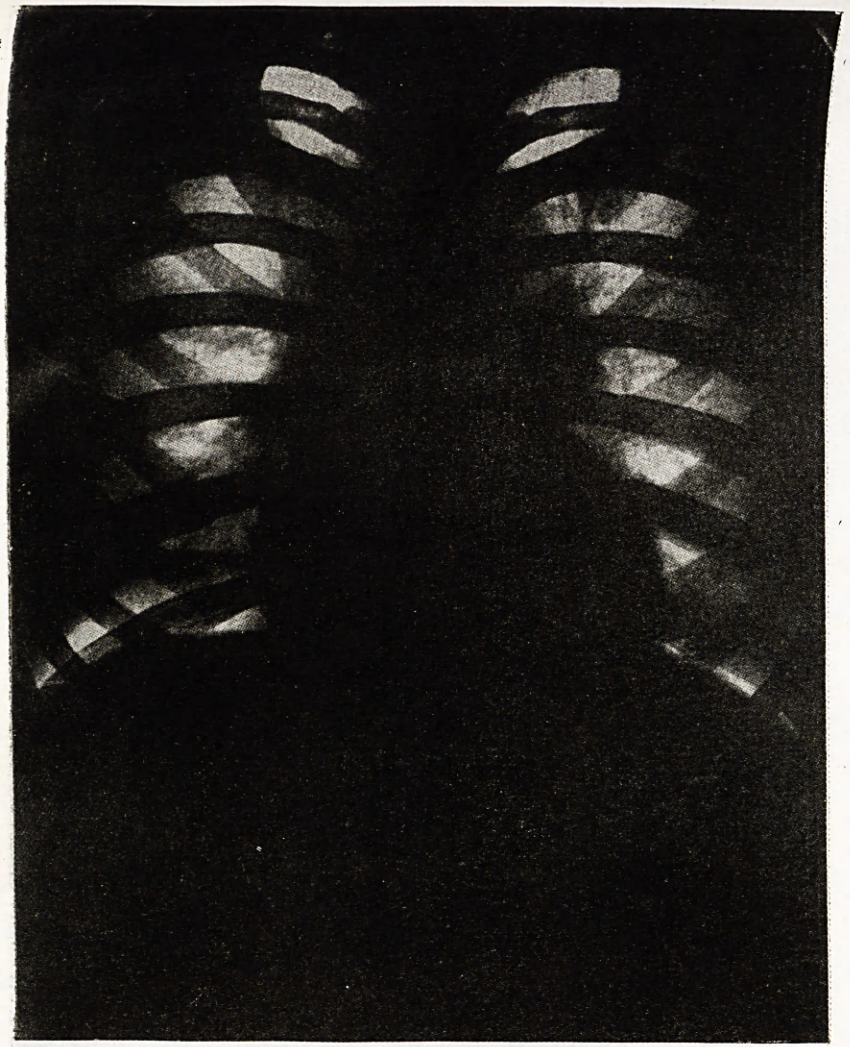


Fig. 2.—(*b*) A.P. view after pneumoperitoneum.

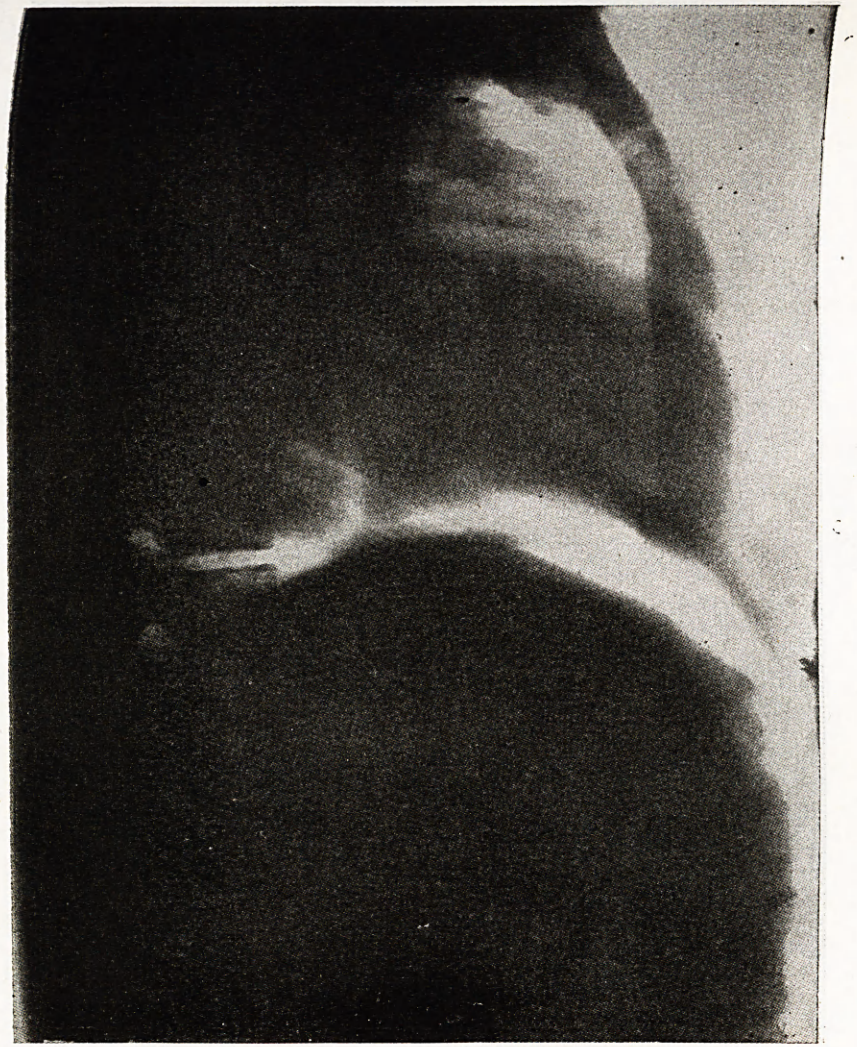
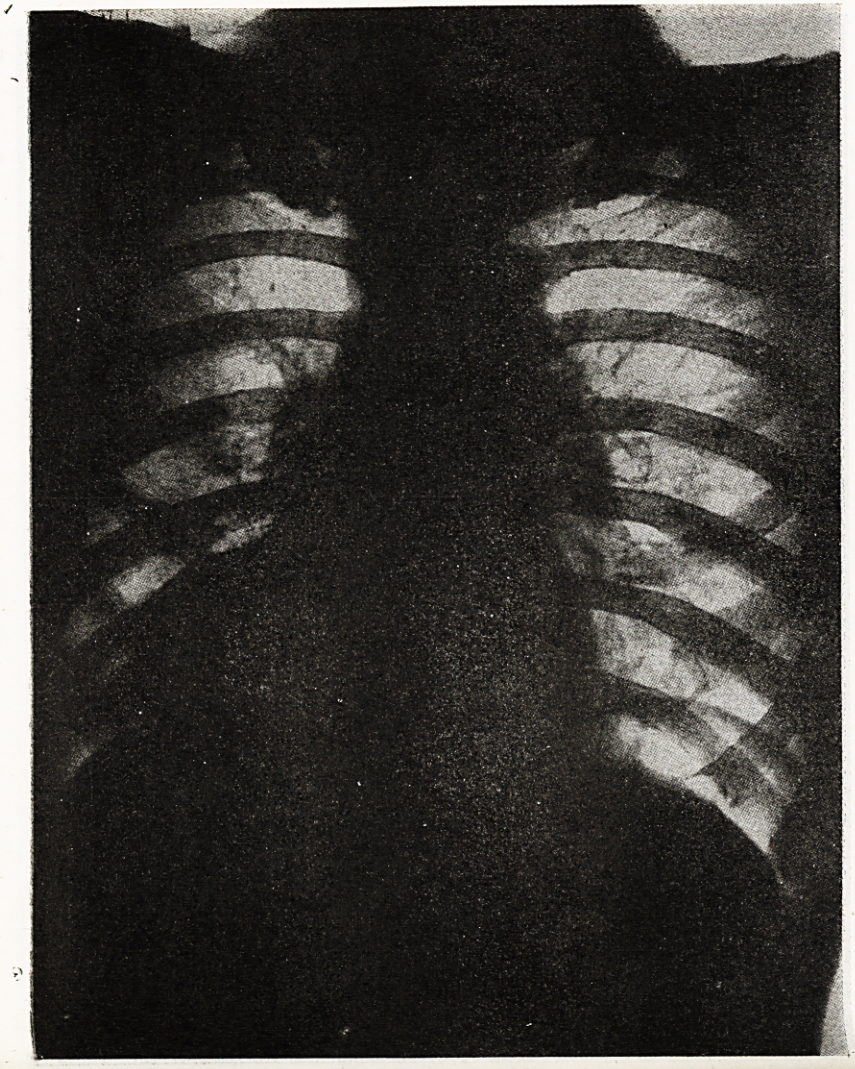


Fig. 2.—(*c*) Lateral view after pneumoperitoneum.

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