THORACIC AMOEBIASIS

A CLINICAL STUDY

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CONTENTS

INTRODUCTION

"Amoebiasis in the African in Durban is a very grave problem". The above statement was made by Kisdon-Dew (1946) 18 years ago and during this period much has been written concerning intestinal and hepatic amoebiasis in our African population. Unfortunately amoebiasis in the African in Durban is still a problem and it is with the thoracic presentation of this problem that this thesis concerns itself.

The term "thoracic amoebiasis" includes all conditions within the thorax caused by the Entamoeba histolytica. Judging from published reports and a considerable personal experience clinicians who see thoracic amoebiasis are still faced with many problems. In this study an attempt will be made to examine some of these problems as they are seen in patients with pleuropulmonary and pericardial amoebiasis at this hospital.

The first part of this study consists of an analysis of the clinical findings, the investigations, the management and the progress of 50 patients with pleuropulmonary amoebiasis. This is followed by a brief review of literature concerning pleuropulmonary amoebiasis and a discussion of the findings in this study.

The second part of this study consists of an analysis of the clinical findings, the investigations, the management and the progress

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of 15 patients with amoebic pericarditis. This is followed by a brief review of the literature concerning amoebic pericarditis and a discussion of the findings in this study.

The 65 patients observed in this study were African males of Zulu extraction and the group as a whole typified African male patients seen at this hospital. The large majority had received very little formal education, were members of the lowest income group and earned their livelihood as unskilled labourers. The cultural background of most patients was such that many had more faith in the "Nyanga" or witch doctor than the medical services available and this frequently resulted in a considerable delay in seeking medical attention.

Patients were admitted to beds in the division of medicine at this hospital during the period January 1960 to December 1962. The criteria for inclusion in this study was a diagnosis of thoracic amoebiasis. Unfortunately, shortage of hospital beds prevented adequate study of all patients admitted with thoracic amoebiasis during the above period. The pleuropulmonary group, however, is considered completely representative of pleuropulmonary amoebiasis as seen at this hospital. A relatively larger number of patients with pericardial than with pleuropulmonary amoebiasis have been included in this study as pericardial amoebiasis is rare, and to date no personal series of any size has been reported in the literature.

All the patients were seen by me shortly after admission and

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the relevant details of their history, past history and their clinical findings were recorded. A record was kept of their response to treatment and clinical progress. When available patients were seen at follow-up clinic.

Relevant investigations were included in the case reports. In each instance the haemoglobin and the white cell count were estimated on admission and at discharge. Sedimentation rates were obtained and in a number of patients the differential count was requested. When anaemia was present the mean corpuscular haemoglobin concentration was estimated and a peripheral blood smear examined. In a few patients the bone marrow was examined and stained for iron and the serum iron and total plasma iron binding capacity estimated. Haematological investigations were done by the routine laboratory at this hospital.

Posterior-anterior and lateral radiographs were obtained on admission, during hospitalisation, at discharge, and at follow-up. Other radiological facilities employed were screening of the chest, tomography, and bronchography.

Sputum, pleural, pericardial and liver aspirates were examined directly for Entamoeba histolytica and in many instances culture of this parasite was attempted. The sputum, pleural and pericardial aspirates were examined and cultured for pyogenic bacteria and acid-fast bacilli. Examination of the sputum and aspirates was done in

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either the routine laberatory at this hospital or in the laberateries of the Amoebiasis Research Unit which has a close association with this hospital.

The stools of each patient were examined for trophozoites and cysts of Entamoeba histolytica.

PART I - PLEUROPULMONARY AMOEBIASIS

THE FINDINGS IN 30 PATIENTS WITH PULMONARY AMOEBIASIS

CLINICAL FINDINGS (Cases 1 - 30)

Age: The study was confined to adult males. The youngest patient was 19 years (case 17) and the eldest 59 years (case 1). The average age for the group was 34 years and 21 patients were within the 3rd and 4th decades (see figure 1).

Duration of Symptoms: The shortest history was of 2 days (case 9), the longest of one year's duration (case 20) with an average duration for the group of 9 weeks. In 19 patients symptoms had been present for one month or less.

Main Complaints: These have been recorded in table 1. Major presenting complaints were pleuritic pain in the right lower chest (27 patients) and a cough productive of reddish-brown sputum or haemoptysis (23 patients). Five patients (cases 10,13,15,22 and 24) who did not complain of a cough productive of reddish-brown sputum developed this symptom following admission. Increased coughing was experienced by 29 patients and the quantity of sputum said to have been coughed varied from several cupfuls to less than a quarter of a cupful per day.

Symptoms suggestive of associated right subphrenic pathology were pain in the right upper quadrant of the abdomen (9 patients), and right shoulder tip pain (14 patients). One patient

/(case 6 ...

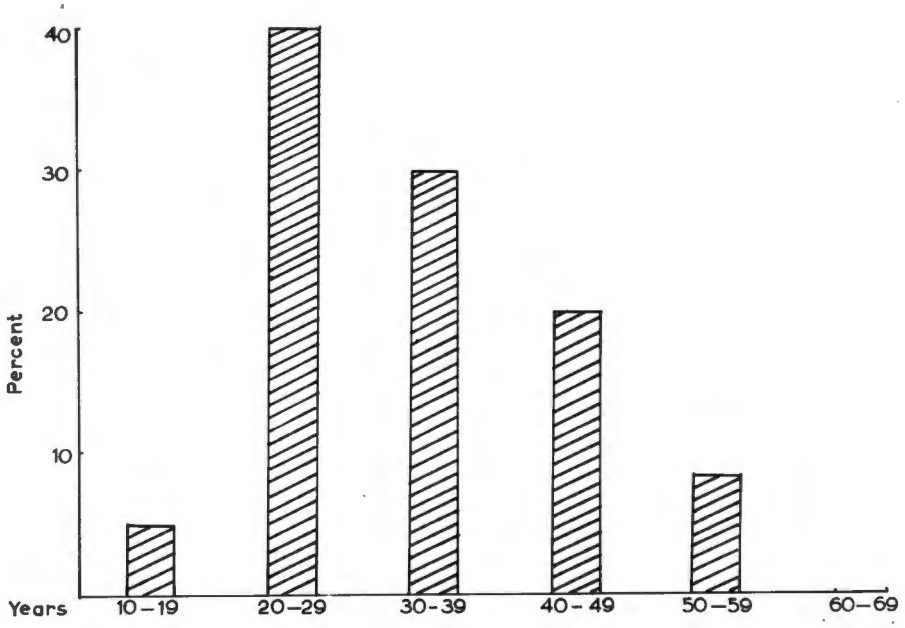


FIG. 1. AGE DISTRIBUTION OF 30 PATIENTS WITH PULMONARY AMOEBIASIS. (CASES 1-30)

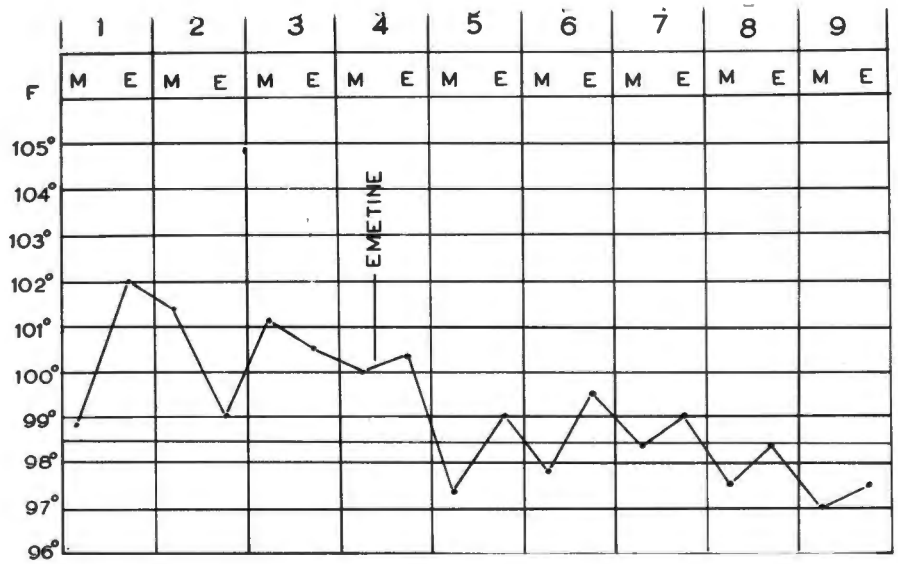


FIG. 2. TEMPERATURE CHART OF PATIENT WITH PULMONARY AMOEBIASIS SHOWING NON-SPECIFIC TYPE OF FEVER AND RESPONSE TO EMETINE. CASE 15.

TABLE I

CLINICAL FEATURES OF 30 PATIENTS WITH PULMONARY AMOEBIASIS

Symptoms	Patients	Per Cent
Pain right lower chest	27	90
Cough	29	97
Haemoptysis or reddish-brown sputum	23	77
Right shoulder tip pain	14	47
Pain right upper quadrant of the abdomen	9	30
Past history of dysentery	8	27
Concomitant dysentery	2	7
Signs		
Signs in the right lower chest	28	93
Cough productive of blood, blood-stained pus or anchovy sauce pus	27	90
Pyrexia	26	87
Guarding and tenderness right upper quadrant of the abdomen	20	67
Localised intercostal tenderness	20	67
Pale mucosa	18	60
Tender hepatomegaly	15	50
Clubbing of the fingers	9	30
Evidence of weight loss	7	23

(case 6) complained of pain in the left upper quadrant of the abdomen.

Two patients (cases 2 and 3) complained of diarrhoea with blood and mucus and 8 admitted to a past history of dysentery. One patient had been treated previously for an amoebic liver abscess (case 18).

General Examination: Pyrexia (26 patients) was generally of a low grade non-specific type. Twenty-three patients were pyrexial on admission, in 19 the temperature remained below 101°F. Four patients (cases 7,17,19 and 30) were apyrexial throughout their stay. (See figure 2).

Pallor of the mucous membranes (18 patients), evidence of weight loss or wasting (7 patients) and clubbing of the fingers (9 patients) were the main findings noted on general examination.

Local Examination: The majority (27 patients) coughed up several hundred ml. of sputum in the early stages of their illness. Those with a non-productive cough usually volunteered that they had coughed up large quantities of sputum at some stage prior to admission. The character of the sputum was either pure blood or blood-stained pus (13 patients) or "anchovy sauce" pus* (14 patients). One patient (case 29) coughed up yellow pus and 2 patients (cases 3 and 26) had a non-productive cough.

* Pus similar to anchovy sauce in colour which in the following pages will be referred to as anchovy sauce pus.

Twenty-eight patients presented with signs in the right lower chest and one patient (case 6) with signs in the left lower chest. In the remaining patient (case 24) there were no chest signs.

Signs suggestive of elevation of the right diaphragm or a pleural effusion were more frequently found than those suggestive of parenchymal involvement. Twenty-eight patients had diminished movement, dullness on percussion, and decreased air entry at the right base whereas only 14 had crepitations and bronchial breathing in this situation. In one patient (case 3) with signs at the right lung base crepitations were heard over the left upper chest and subsequently at necropsy multiple small abscesses were found in the left upper lobe.

One patient (case 29) who coughed purulent sputum had signs of a large effusion in the right lower chest, which clinically suggested pleural as well as pulmonary involvement.

Signs suggestive of associated subphrenic pathology were guarding and tenderness in the right upper quadrant (20 patients), localised intercostal tenderness (20 patients) and a tender enlarged liver (15 patients).

HAEMATOLOGICAL FINDINGS

Haemoglobin: The haemoglobin levels and distribution for the group have been recorded in table 2. In 23 patients the haemoglobin level was less than 12 g. per cent, and in 13 of these the anaemia was

/severe ...

TABLE 2

HAEMATOLOGICAL FINDINGS IN 30 PATIENTS WITH PULMONARY ANEURYSMS

Hb g. per cent	No. of Patients	Per Cent
12 - 15	7	23
9 - 11.9	10	33
< 9	13	43

W.B.C. per c.mm.	No. of Patients	Per Cent
< - 10,000	8	27
10,100 - 20,000	19	63
> 20,000	3	10

severe with haemoglobin levels of less than 9 g. per cent.

In 22 of the 23 patients with anaemia, examination of peripheral smears and the mean corpuscular haemoglobin concentration (M.C.H.C.) estimation indicated that the anaemia was normocytic normochromic in type. In the remaining patient (case 6) the peripheral smear showed a microcytic hypochromic picture and the M.C.H.C. was 25 per cent.

Estimation of the serum iron and total iron binding capacity of the plasma and examination of the bone marrow for iron in 4 patients showed findings similar to those reported in the anaemia of infection.

White Cell Count: Details of the white cell counts have been recorded in table 2. In 8 patients the white cell count was 10,000 per c.mm. or less, whilst in the remaining 22, counts of greater than 10,000 per c.mm. were obtained. Only 3 patients (cases 8,13 and 15) had white cell counts of greater than 20,000 per c.mm.

Sedimentation Rate: The blood sedimentation rate was estimated in 28 of the group on admission, using Wintrobe's method. It was increased in each of these patients with a range from 20 mm. (case 17) to 84 mm. in one hour (case 3), and an average for the group of 56 mm. in one hour.

RADIOLOGICAL FINDINGS (See plates 1 - 5)

Table 3 records the details of the radiological findings. It has been divided into three parts. Part one records the details of the pulmonary changes seen on admission, part 2 the subsequent pulmonary changes, and part 3 the associated diaphragmatic and pleural changes. A striking feature of these radiological changes was the frequency with which they were confined to the base of the right lung.

Cavitation: Twelve patients presented with or developed cavitation. Cavitation of the right lower lobe was seen in the initial films of 6 patients (cases 1,2,5,7,8 and 14). In 5 the cavity was situated in the basal aspect adjacent to the diaphragm and in one (case 2) in the apical segment.

A further 5 patients who initially presented with consolidation of the right lower lobe subsequently developed cavitation. Four of these cavities (cases 4,9,11 and 12) were situated in the basal aspect of the right lower lobe and one (case 13) in the apical segment of the right lower lobe. One patient (case 10) whose films showed no pulmonary changes initially, subsequently developed a cavity in the basal aspect of the right lower lobe.

Associated elevation of the right diaphragm was seen in 10 of the 12 patients with cavitation.

Consolidation: Twenty patients presented with infiltration or

/consolidation ...

TABLE 3

RADIOLOGICAL FINDINGS IN THE PULMONARY GROUP

A. Presenting Pulmonary Changes

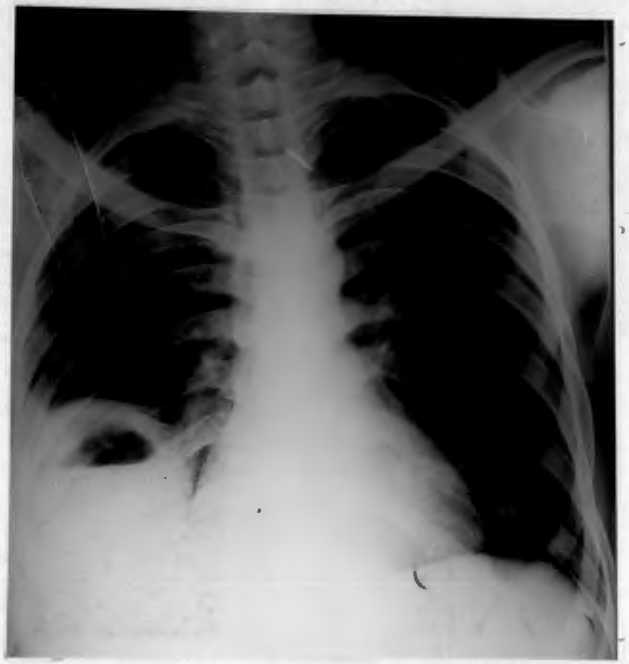
Cavitation right lower lobe	Basal	5
	Apical	1
Consolidation		
- Circumscribed right basal		8
- Lobar (right lower and or middle)		7
- Patchy (right lower lobe)		5
- Left lower lobe		1
No parenchymal changes		3
	Total	30

B. Subsequent Pulmonary Changes

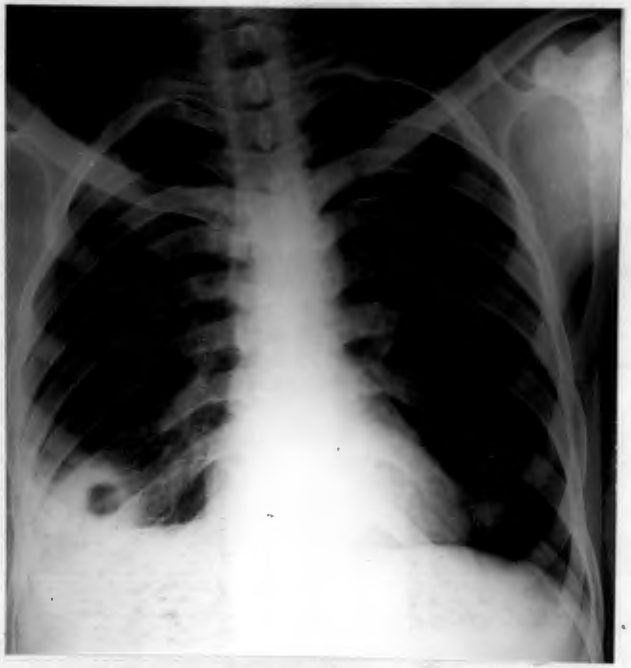
Consolidation progressing to cavitation	
- Circumscribed basal	3
- Lobar (right lower)	1
- Patchy (right lower lobe)	1
No change initially with subsequent cavitation	1
Total	6

C. Associated Changes

Elevation of the right hemidiaphragm		23
Air-fluid level beneath the right diaphragm		3
Small pleural effusions - right base	Interlobar	4
	Costophrenic	4
- left base		1



(a) Before treatment



(b) After 10 days of emetine, chloroquine and postural drainage

Plate 1 Case 1 Postero-anterior radiographs of the chest of a patient with a hepatobronchial fistula and amoebic lung abscess.

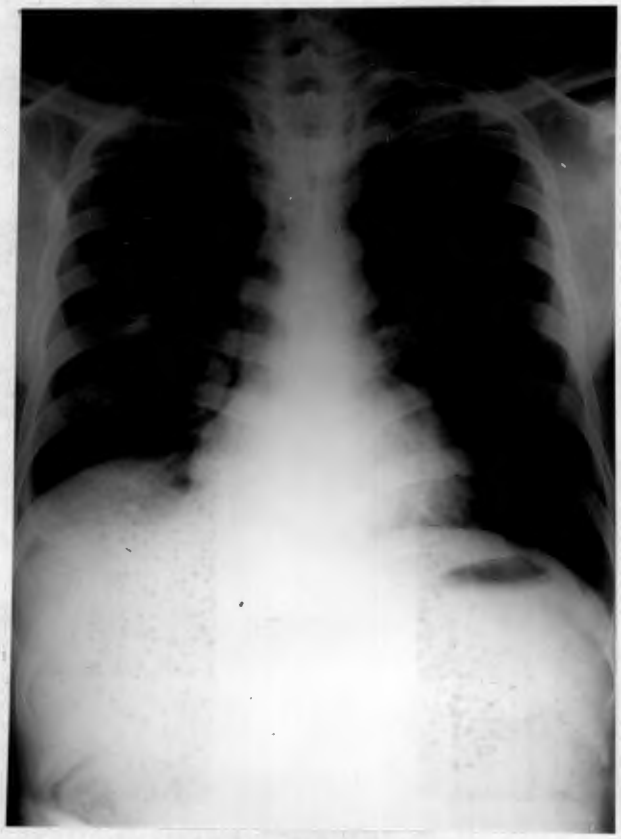
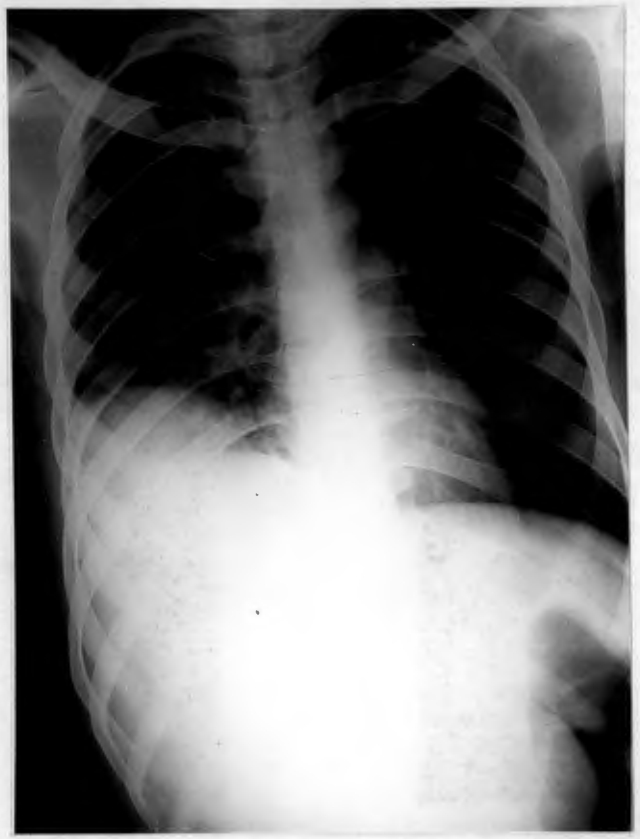
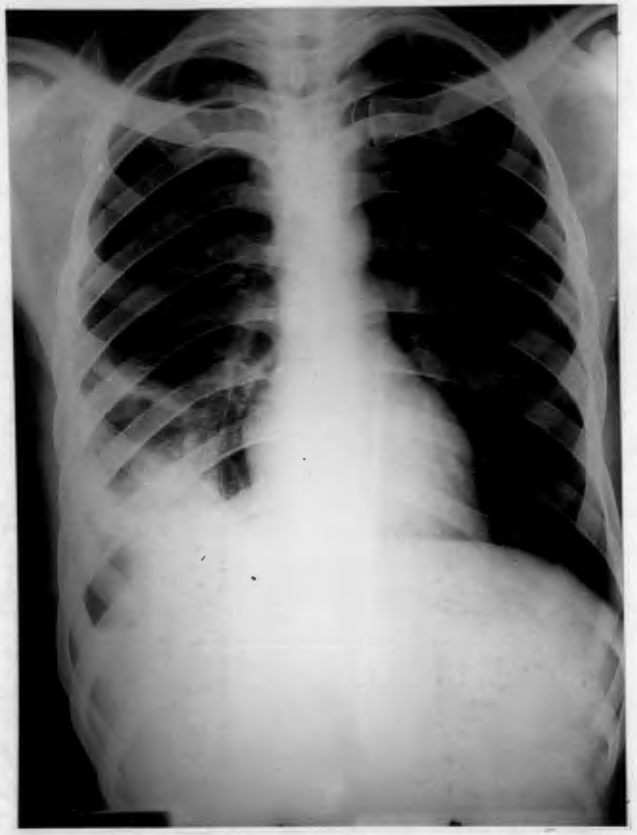


Plate 2 Case 6 Postero-anterior radiograph of the chest showing an area of circumscribed semi-circular consolidation adjacent to the right diaphragm in a patient with a hepatobronchial fistula.

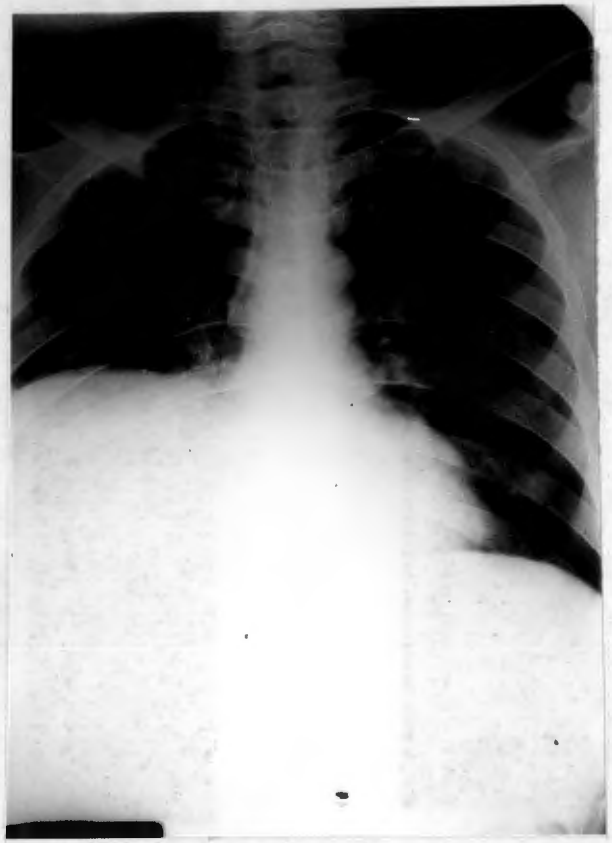


(a) An area of semicircular consolidation can be seen adjacent to the right diaphragm.

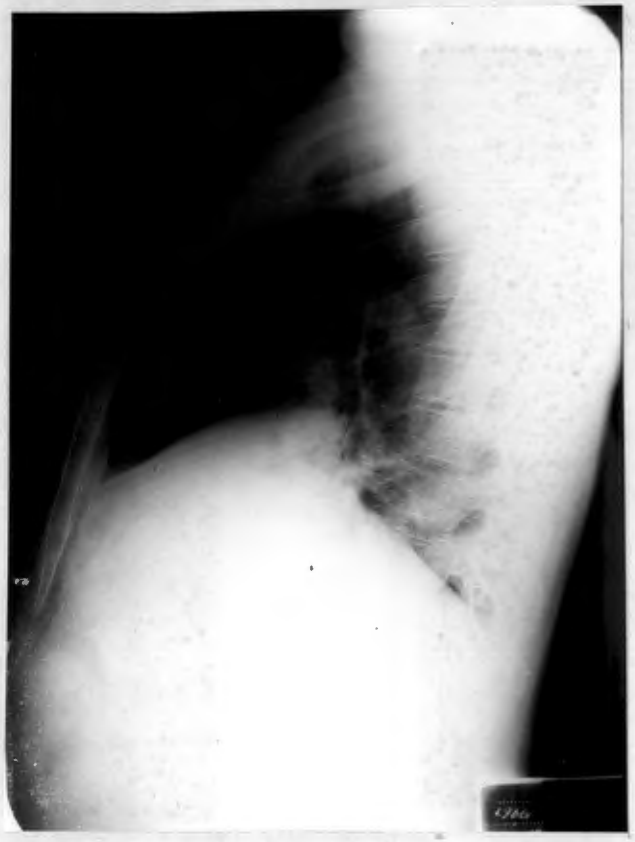


(b) The consolidation has been replaced by an abscess cavity. Air with a fluid level can be seen in the liver abscess.

Plate 3 Case 12 Postero-anterior radiographs of the chest showing replacement of consolidation by cavitation in a patient with a hepatobronchial fistula.

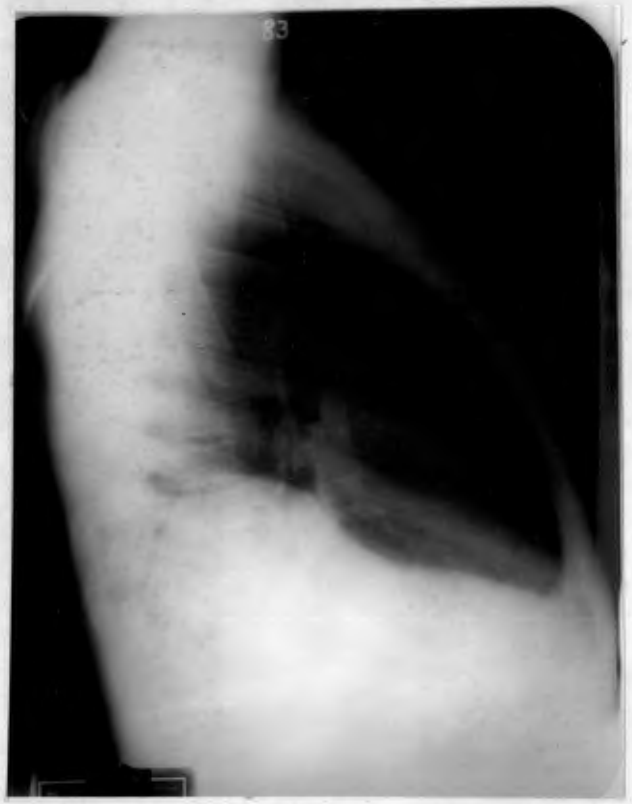


(a) Postero-anterior view

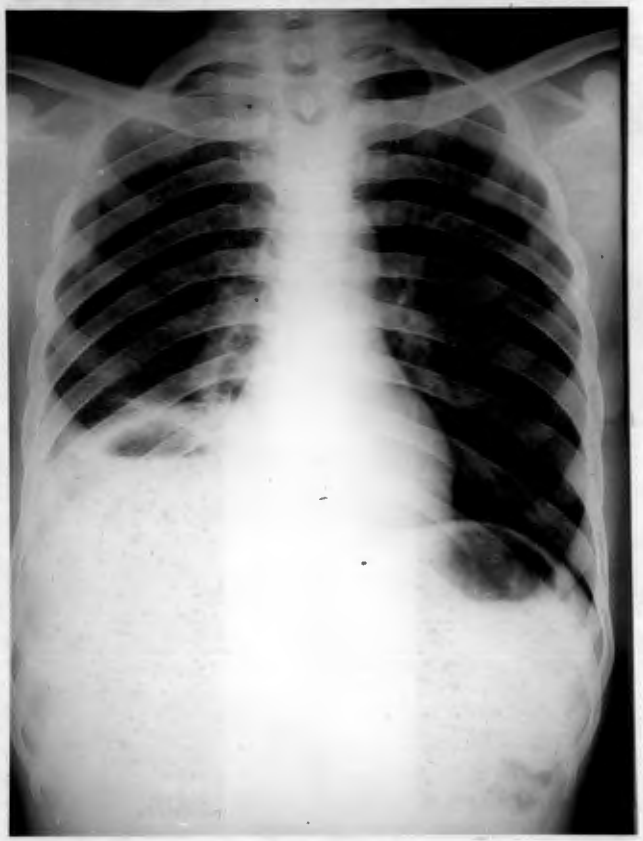


(b) Lateral view

Plate 4 Case 21 Radiographs of the chest showing elevation and deformity of the right diaphragm in a patient with a hepatobronchial fistula.



Case 8 Lateral radiograph of the chest showing localised posterior elevation of the right diaphragm



Case 28 Postero-anterior radiographs of the chest showing an air-fluid level below an elevated right diaphragm

Plate 5 Radiological changes seen with a hepatobronchial fistula

consolidation at the right base. In 18 of these the changes were confined to the right lower and or middle lobes. In one (case 3) there was associated patchy consolidation in the left upper lobe, which was subsequently shown at necropsy to represent multiple small lung abscesses in this situation. Only one patient (case 6) presented with consolidation at the left base. Three varieties of consolidation or infiltration were noted.

(i) In 8 of these 20 patients (cases 4,11,12,15,17,20,24 and 26) the consolidation took the form of circumscribed semicircular infiltration of the basal aspect of the right lower lobe, with the base of the semicircle formed by the right hemidiaphragm. Three of these (cases 4,11 and 12) subsequently developed cavitation at this site.

(ii) Seven of these patients (cases 3,9,18,19,23,25, and 30) presented with lobar consolidation of the right middle and or lower lobes and one of these (case 9) subsequently developed a cavity in the basal aspect of the right lower lobe.

(iii) Patchy consolidation or infiltration of the right lower lobe was seen in 5 patients (cases 13,21,27,28 and 29). One of these patients (case 13) subsequently developed a cavity in the apical segment of the right lower lobe. A further patient (case 6) presented with patchy consolidation in the basal aspect of the left lower lobe.

Three patients (cases 10,16 and 22) had no radiological pulmonary changes on admission. One of these (case 10) subsequently developed a cavity in the basal aspect of the right lower lobe.

Elevation of the Diaphragm: Twenty-three of the 30 patients showed associated generalised or localised elevation of the right diaphragm which probably contributed in some instances to the parenchymal changes at the right base. Three patients (cases 21,28 and 29) were seen to have air-fluid levels below the right diaphragm before liver aspiration was attempted.

Pleural Effusions: Eight patients were seen to have associated pleural effusions. Four of these (cases 9,12,26 and 30) were small inter-lobar effusions spreading the base of the greater fissure, three (cases 13,14 and 22) were small effusions situated in the right costo-phrenic angle and one (case 29) was a large effusion in the right costo-phrenic angle with air-fluid levels. A further patient (case 6) subsequently developed a large effusion at the left base.

Screening: The chest was screened in 4 patients. Three (cases 1,5 and 29) showed elevation and immobility of the right diaphragm and one (case 6) similar changes on the left side. In this last patient (case 6) a filling defect or indentation of the greater curvature of the barium filled stomach was seen on screening.

Bronchography: Bronchograms were obtained in 5 patients and evidence

/of ...

of bronchiectasis of the right middle or lower lobe was found in 3 (cases 2,23 and 29). In one patient (case 23) crowding of the basal bronchi of the right lower lobe suggested atelectasis.

SPUTUM EXAMINATION

Trophozoites of E.histolytica were seen on direct examination of the sputum of 10 patients (cases 1,4,5,8,9,14,15,21,23 and 30). Attempted culture of the parasite was nearly always unsuccessful, even of sputum in which it was identified on direct examination.

Acid-fast bacilli were not identified on direct examination or culture of the sputum. In a number of patients light to moderate growths of normal respiratory flora were reported on culture, and in 6 instances (cases 2,9,23,25 and 29) moderate to heavy growths of organisms such as Staphylococcus pyogenes, Bacillus coli, and Klebsiella pneumoniae were reported.

LIVER ASPIRATION

Liver aspiration was attempted in 16 patients with success in 9. Quantities of pus obtained at the 1st aspiration varied from 5 ml. (case 11) to 500 ml. (case 6). For purposes of drainage a 2nd aspiration was required in 2 patients (cases 10 and 22) and a 3rd aspiration in one (case 10). The aspirate of 6 patients (cases 2,6,21, 24 and 26) was typical anchovy sauce pus and in 3 (cases 3,10 and 11) blood-stained yellow pus was obtained. Trophozoites of E.histolytica were seen on direct examination in the pus of 3 patients (cases 3, 6

/and ...

and 10). The liver pus was sterile on bacterial culture in each of the 9 patients.

PLEURAL ASPIRATION

Small quantities of serosanguineous fluid were aspirated from the right pleural cavity of 2 patients (cases 13 and 22) and serous fluid from one (case 14). These effusions were exudates, contained a predominance of red blood cells and or polymorphonuclear leucocytes, and acid-fast bacteria and pyogenic organisms were absent on direct examination and culture.

Five aspirations yielded a total of one and a half litres of anchovy sauce pus from the right pleural cavity of one patient (case 29) included in this group as an example of right basal pulmonary involvement. Trophozoites of E.histolytica were identified in the pus on direct examination and Staph.pyogenes, B.coli and Proteus vulgaris cultured from aspirates.

STOOL EXAMINATION

Trophozoites of E.histolytica were identified in the stools of 4 patients (cases 2,3,5 and 21), and cysts of E.histolytica in the stools of one patient (case 30).

TREATMENT

Twenty-seven patients received the tissue amoebicides, emetine hydrochloride and chloroquine (phosphate or sulphate) with the intestinal

/amoebicide ...

amoebicide diiodohydroxyquinoline in the following doses:

(i) Emetine hydrochloride gr. 1 intramuscularly for 10 days.

(ii) Chloroquine (phosphate or sulphate) 600 mg. of the base immediately, 300 mg. 6 hours later and then 150 mg. twice daily for 27 days.

(iii) Diiodohydroxyquinoline 600 mg. thrice daily for 21 days.

In 3 patients (cases 5,23 and 14) the synthetic analogue of emetine, dehydroemetine was substituted for emetine hydrochloride in the above regime. The dosage used was 80 mg. intramuscularly daily for 10 days.

Twelve patients received specific treatment from the 1st day, 8 from the 2nd day, 5 from the 3rd day and a further 4 within the 1st week. In one instance (case 6) there was a delay of 6 weeks before the diagnosis was made and specific therapy started.

Fifteen patients received only amoebicidal drugs in the form of emetine hydrochloride, chloroquine and diiodohydroxyquinoline and the remaining 15 received one or more antibiotics in addition to specific treatment. Blood transfusions were administered in 3 instances (cases 3, 6 and 29).

Drainage Procedures: These consisted of postural drainage, liver aspiration and pleural aspiration when indicated. The details of the liver and pleural aspirations have been recorded.

The indications for liver aspiration and the optimal sites chosen for aspiration were essentially similar to those proposed by Wilmot (1962). A localised swelling or bulging of the ribs, marked localised tenderness or oedema and clinical and radiological evidence of an elevated diaphragm, were findings regarded as indications. If more than 200 ml. of pus was obtained at aspiration the procedure was usually repeated within 2 to 3 days' time.

CLINICAL PROGRESS

Table 4 records the details of progress in the 30 patients with pulmonary amoebiasis. There were 2 deaths and 28 recoveries. There was no apparent residual disability in any of the 28 recoveries and each of these patients was able to pursue his normal occupation. The duration of hospitalisation for these patients ranged from 10 to 75 days with an average of 29 days.

The temperature settled on an average of 8 days following the commencement of treatment in the 24 survivors who were pyrexial on admission. Four patients remained apyrexial throughout their stay (cases 7,17,19 and 30).

There was an average weight increase of 8 lbs. per patient during hospitalisation in the 24 patients in whom this was recorded.

Significant sputum production ceased on an average of 12 days following the onset of specific treatment in the 27 survivors with a productive cough. The majority of patients coughed up several

/hundred ...

TABLE 4

CLINICAL PROGRESS OF 30 PATIENTS WITH PULMONARY AMOEBIASIS

Observations		Total No. of Patients Observed	Per Cent
Deaths	2	30	12
Average duration of pyrexia (in days after treatment)	8	24	-
Average duration of hospitalisation (days)	29	28	-
Average weight increase in lbs.	8	24	-
Sputum production average duration days after treatment	12	27	-
Signs at the right lung base at discharge	21	28	75
Resolution of abdominal tenderness	20	20	100
Absence of hepatomegaly at discharge	13	14	93
Follow-up progress maintained	7	7	100

hundred millilitres of anchovy sauce, blood or blood-stained pus within the first few days and thereafter decreasing quantities from about 300 ml. to 50 ml. were coughed up daily until sputum production ceased. In most instances the character of the sputum gradually changed from pus to a mucoid sputum.

At discharge 21 patients had residual signs at the right base suggestive of elevation of the right hemidiaphragm. There was complete clearing in every instance of signs suggestive of consolidation and abscess formation.

Right upper quadrant guarding and tenderness resolved in all survivors, frequently within the 1st week following treatment. A firm non tender enlarged liver persisted in only one (case 15) of the 14 survivors who presented with a tender hepatomegaly.

Seven patients (cases 2,4,8,11,17,18 and 22) attended follow-up clinic regularly for periods of one to 21 months. In each of these clinical progress was maintained.

HAEMATOLOGICAL PROGRESS

Haemoglobin estimations at discharge in 25 of the 28 survivors showed that there had been an average rise of 2.2 g. per patient during hospitalisation. Blood transfusions were administered to 3 patients but in the majority the anaemia responded following treatment with emetine and chloroquine.

The white cell count at discharge was 10,000 per c.mm. or

/less ...

loss in 16 of the 20 survivors in whom it was raised on admission.

There was a significant decrease in the sedimentation rate in each of the 15 survivors in whom serial estimations were obtained.

RADIOLOGICAL PROGRESS

Table 5 summarises the details of radiological progress in 26 of the 28 survivors who presented with or subsequently developed radiological changes.

Cavitation: There was complete healing in 10 (cases 1,2,4,5,8,10, 11,12,13, and 14) of the 12 patients who presented with or developed cavitation of the right lower lobe. Two patients (cases 7 and 9) who failed to attend follow-up clinic had small residual cavities at discharge.

Consolidation: There was satisfactory clearing of consolidation or infiltration at the right base in each of the 26 survivors who presented with or developed these changes. Serial films in 8 patients (cases 2,11,14,17,20,25,27 and 29) showed complete clearing of right basal changes. In 18 patients small residual shadows or minimal patchy infiltration persisted at the right base.

Elevation of the Right Diaphragm: Of the 23 patients who presented with elevation of the right hemidiaphragm this persisted at discharge in 17. In 5 patients (cases 7,14,15,20 and 30) the right hemidiaphragm had returned to normal position. Air-fluid levels below the right diaphragm cleared in each of the 3 patients (cases 21,28,29) in whom

/these ...

TABLE 5

RADIOLOGICAL FINDINGS OF 26 PATIENTS WITH PULMONARY ABSCESSES

Observations		Total No. of Patients Observed	Per Cent
Healing of abscess cavities	10	12	83
Complete clearing of right basal shadows	8	26	33
Minimal persistent shadows right base	18	26	67
Residual elevation of the right diaphragm	17	23	74
Clearing of subphrenic air-fluid levels	3	3	100
Resolution of associated effusions	9	9	100

these were observed.

Pleural Effusions: There was complete resolution of the pleural effusions in each of the 9 patients (cases 9,12,13,14,19,22,26,29 and 30) in whom these were associated findings.

NECROPSY FINDINGS (See plate 6)

There were 2 deaths (cases 3 and 6) in this group and permission for necropsy was obtained on both patients. In the first (case 3) a hepatopulmonary amoebic abscess was found at the right base with multiple amoebic lung abscesses in the left upper lobe.

In the second (case 6) an abscess cavity was found in the left lower lobe which communicated through the diaphragm with the stomach. Immediately beneath the left basal lung abscess an area of scar tissue thought to represent the remains of a healed amoebic abscess was found in the left lobe of the liver.

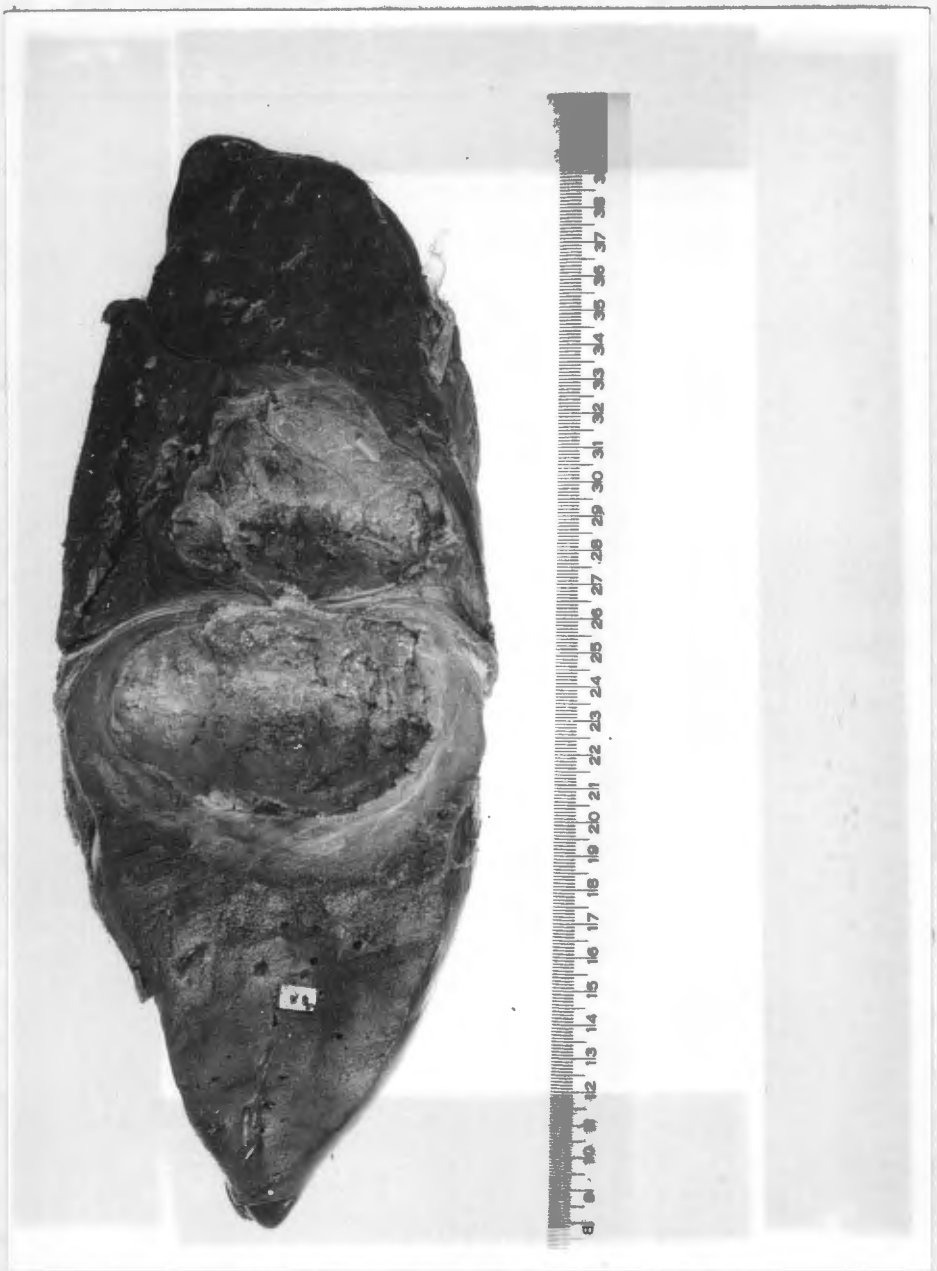


Plate 6 Necropsy findings in hepatopulmonary amoebiasis showing a large amoebic liver abscess communicating through the diaphragm with an amoebic lung abscess in the right lower lobe.
(scale in cms.)

THE FINDINGS IN 20 PATIENTS WITH PLEURAL AMOEBIASIS

CLINICAL FINDINGS

(This group includes 5 patients (cases 31 - 35) with presuppurative amoebic effusions and 15 patients (cases 36 - 50) with suppurative amoebic effusions)

Age: The study was confined to adult males. The youngest patient was 22 years (case 32) and the oldest 60 years (case 31). The average age for the group was 38 years and 10 fell within the 3rd and 4th decades (see figure 3).

Duration of Symptoms: The shortest history was of 3 days (case 48) and the longest of one year's duration (case 47) with an average for the group of 10 weeks. In 11 patients symptoms had been present for a month or less.

Main Complaints: These have been recorded in table 6. Major symptoms were pleuritic pain in the right lower chest (15 patients) and cough (17 patients). Eight patients complained of dyspnoea which was not a prominent symptom in the pulmonary group. Ten patients complained of coughing up blood-stained or reddish-brown sputum, an important symptom in that it was often the only evidence of associated pulmonary involvement. One patient (case 42) volunteered that 16 days before admission he had experienced a "bursting" sensation associated with severe pain in the right lower chest.

Symptoms suggestive of associated subphrenic pathology were

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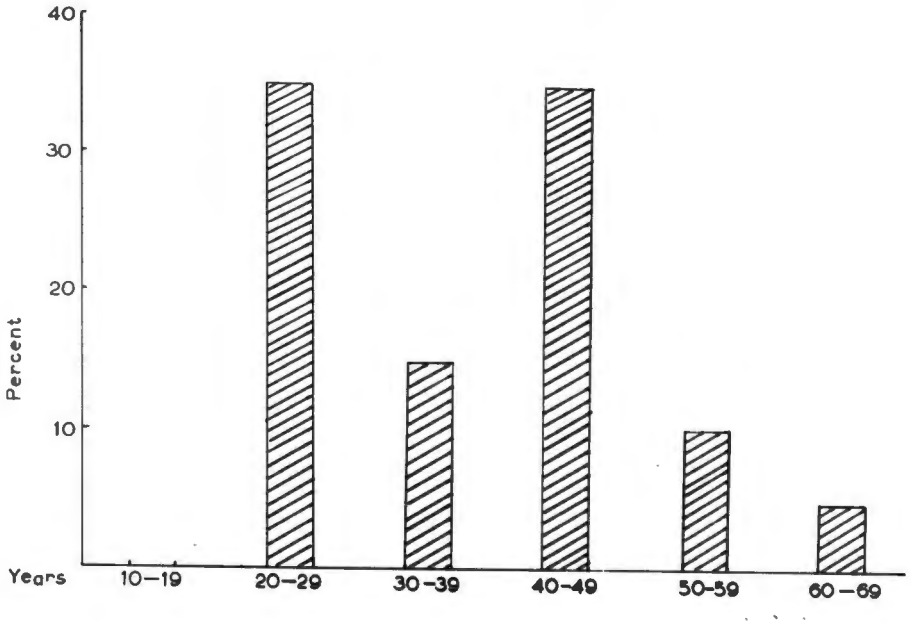


FIG 3. AGE DISTRIBUTION OF 20 PATIENTS WITH PLEURAL AMOEBIASIS. (CASES 31-50)

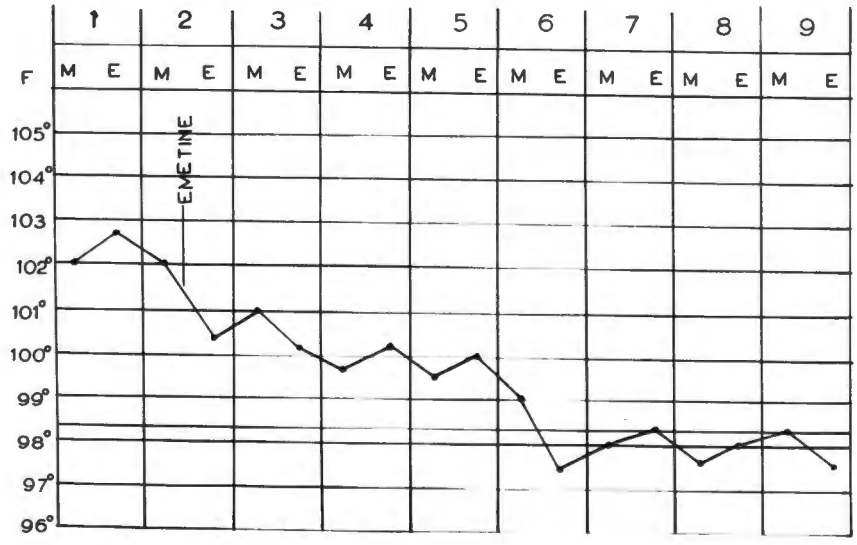


FIG. 4. TEMPERATURE CHART OF PATIENT WITH PLEURAL AMOEBIASIS: CASE 45, SHOWING NON-SPECIFIC TYPE OF FEVER AND RESPONSE TO EMETINE

TABLE 6

CLINICAL FEATURES IN 20 PATIENTS WITH PERINEAL ANCHYLASIS

Symptoms	Patients	Per Cent
Cough	17	85
Pain in the right lower chest	15	75
Pain in the right upper quadrant	10	50
Haemoptysis or reddish-brown sputum	10	50
Dyspnoea	8	40
Right shoulder tip pain	5	25
Past history of dysentery	5	25
Concomitant dysentery	4	20
Local swelling in the right upper quadrant	2	10
Signs		
Pyrexia	17	85
Signs in the right lower chest	17	85
Guarding and tenderness in the right upper quadrant	17	85
Tender hepatomegaly	15	75
Localised intercostal tenderness	14	70
Pale mucosa	14	70
Evidence of weight loss	13	65
Cough productive of pus	8	53

a continuous right upper quadrant pain of variable severity (10 patients) and right shoulder tip or supraclavicular pain (5 patients). A localised swelling in the right upper quadrant had been noticed by 2 patients (cases 31 and 41).

Four patients (cases 33,43,48 and 49) complained of diarrhoea with blood and mucus and 5 (cases 33,34,41,46 and 49) admitted to a past history of dysentery.

General Examination: Seventeen patients were pyrexial on admission. The highest temperature was 103°F., and in 12 patients it remained below 101°F. Two patients (cases 35 and 47) were apyrexial during hospitalisation.(See figure 4).

Pallor of the mucous membranes was detected in 14. Evidence of weight loss or wasting, a finding in 13, was more noticeable than in the pulmonary group, and 4 patients (cases 37,43,47 and 49) were frankly emaciated.

Local Examination: Four patients (cases 32,34,41 and 43) presented with prominent anterior and lateral bulging of the right lower thoracic cage.

Signs suggestive of an effusion were detected at the right base in 17 patients on admission. In the remaining 3 patients signs of fluid developed following hospitalisation. Of these one patient (case 41) suddenly developed signs of a massive effusion in the right chest on the 2nd day, one patient (case 48) developed signs at the right base

/ on ...

on the 15th day and in one patient (case 40) signs were detected at the left base on the 4th day.

It was sometimes impossible to decide whether signs at the right base represented an elevated diaphragm or a small to moderate sized effusion and in a number of patients chest radiographs were required to make this distinction.

Ten patients (cases 36,37,39,42,43,44,45,46,47 and 50) presented with signs in the right chest suggestive of a large pleural effusion. In 6 of these (cases 39,42,44,45,46 and 50) there was evidence of mediastinal shift. A further patient (case 41) developed signs of a large effusion with mediastinal shift on the 2nd day. It can be seen that the patients with signs of a large effusion were in the group with suppurative pleural amoebiasis (cases 36 - 50). In the group with presuppurative amoebic effusions (cases 30 - 35) signs suggestive of fluid were usually confined to the right base suggesting small to moderate sized effusions. It was in this group (cases 30 - 35) that difficulty was experienced clinically in determining whether signs at the right base represented an effusion or elevated right diaphragm.

One patient (case 36) had signs in the right chest of a hydropneumothorax.

Eight patients in the group coughed up quantities of pus, 3 (cases 39,47 and 49) coughed up blood-stained pus, 3 (cases 36, 42 and 50) yellow pus and 2 (cases 32 and 41) typical anchovy sauce

/pus ...

pus. The productive cough in these patients was often the only clinical evidence of associated pulmonary involvement.

Signs suggestive of associated subdiaphragmatic pathology were guarding and tenderness in the right upper quadrant (17 patients), localised intercostal tenderness (14 patients) and a tender enlarged liver (15 patients).

HAEMATOLOGICAL FINDINGS

Haemoglobin: Haemoglobin levels for the group have been recorded in table 7. In 16 of the 20 patients the haemoglobin was less than 12 g. per cent. In 7 patients the anaemia was severe with haemoglobin levels of less than 9 g. per cent. Examination of peripheral smears and estimation of the M.C.H.C. showed the anaemia to be normocytic normochromic in type in 15 of 16 patients with haemoglobin levels of less than 12 g. per cent. In the remaining patient (case 39) there were features suggestive of an iron deficiency anaemia.

White Cell Count: Details of the white cell counts have been recorded in table 7. In 6 patients the white cell count was 10,000 per c.mm. or less, whilst in the remaining 14 counts of more than 10,000 per c.mm. were obtained. Three patients (cases 37, 48 and 50) had counts of greater than 20,000 per c.mm.

Sedimentation Rate: The sedimentation rate was estimated in 19 patients on admission using Wintrobe's method. It was increased in every instance and ranged from 28 mm. in one hour (case 33) to 76 mm. in one hour (case

/37) ...

TABLE 7

HAEMATOLOGICAL FINDINGS IN 20 PATIENTS WITH PLEURAL AMEBIASIS

Hb g. per cent	No. of Patients	Per Cent
12 - 15	4	20
9 - 11.9	9	45
< 9	7	35

W.B.C. per c.mm.	No. of Patients	Per Cent
< 10,000	6	30
10,100-20,000	11	55
> 20,000	3	15

37) with an average for the group of 55 mm. in one hour.

RADIOLOGICAL FINDINGS (See plates 7 and 8)

Chest radiographs were obtained in 19 of the 20 patients in this group. One patient (case 38) died before films could be obtained.

Table 8 records the radiological findings. In 18 of the 19 patients the effusion was situated in the right chest. Fifteen of these were right basal effusions and 3 (cases 44, 45 and 47) were encysted. In 15 of the 18 patients with right sided effusions the effusion was present on admission while in 3 (cases 41,43 and 48) it developed during hospitalisation. The remaining patient (case 40) developed a left sided basal effusion on the 7th day. A further patient (case 48) with a right sided effusion subsequently developed a small left sided effusion.

The patients in the presuppurative group (cases 31 - 35) were seen to have effusions which were small to moderate in size. Those with amoebic empyema, i.e. the suppurative group (cases 36 - 50), usually presented with large sometimes massive effusions. In 9 (cases 36,37,39,41,42,44,45,46 and 50) of the 15 patients in the suppurative group mediastinal shift was observed.

Air-fluid levels were seen in the pleural space in 5 patients (cases 36,41,42,47 and 50) in the suppurative group. Four of these (cases 36,41,47 and 50) were considered on the basis of

/clinical ...

TABLE 8

RADIOLOGICAL FINDINGS IN 19 PATIENTS WITH PLEURAL AMOEBIASIS

Right Basal Effusion	15
Right Encysted Effusion	3
Left Basal Effusion	1
Air-Fluid Levels Pleural Cavity	5
Elevated Right Hemidiaphragm	14
Elevated Left Hemidiaphragm	1
Associated Parenchymal Changes	
- Cavitation Right Lower Lobe	1
- Consolidation Right Lower Lobe	2
- Consolidation Right Middle and Lower Lobes	1

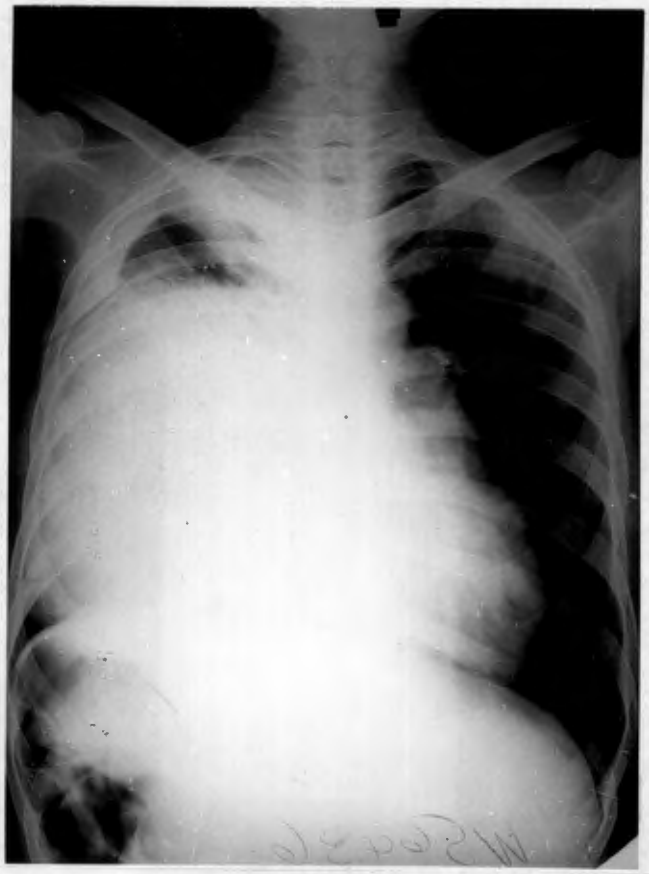
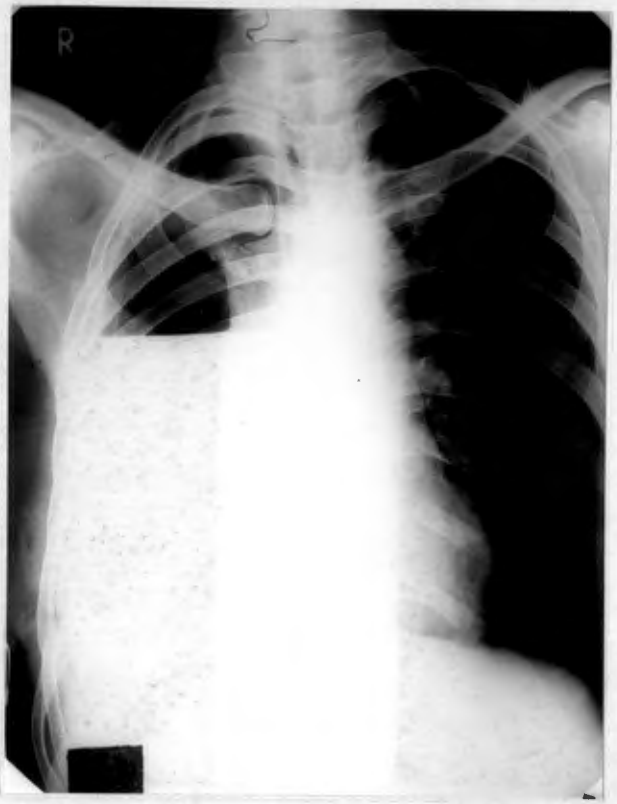
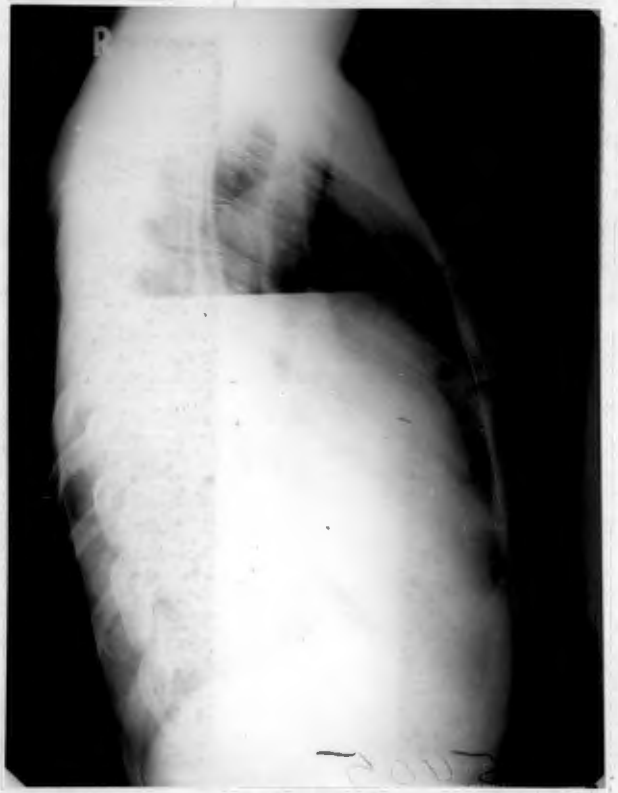


Plate 7 Case 46 Postero-anterior radiograph showing a large right sided effusion in a patient with an amoebic empyema.



(a) Postero-anterior view showing a large right effusion with an air-fluid level



(b) Lateral view

Plate 8 Case 41 Radiographs of the chest of a patient with an amoebic empyema and bronchopleural fistula

clinical and radiological findings to have bronchopleural fistulae. In the remaining patient (case 42) the air was probably introduced on aspiration of the chest.

Elevation of the Diaphragm: Initial radiographs showed the right diaphragm to be elevated in 10 patients (cases 31,32,34,35,41,42, 43,45,48 and 49) and in normal position in one (case 37). In the remaining 7 patients with right basal effusions (cases 33,36,39,44, 46,47 and 50) its position was obscured in the films by the pleural fluid. With clearing of the effusions, subsequent radiographs showed the right diaphragm to be elevated in 4 (cases 33,36,39 and 47) of these 7 patients. The left diaphragm was elevated in the patient (case 40) with the left basal effusion.

Parenchymal Changes: Associated parenchymal changes were present in 4 patients. One patient (case 32) had cavitation in the right lower lobe, a further 2 (cases 41 and 49) consolidation of the right lower lobe and the remaining patient (case 39) consolidation with cystic changes of the right middle and lower lobes.

Chest Screening: Three patients had their chests screened and in 2 of these (cases 33 and 50) there was elevation and immobility of the right hemidiaphragm.

Bronchography: Bronchograms were obtained in 2 patients, in one (case 42) there was distortion and partial atelectasis of the right

/lower ...

lower and middle lobes as a result of gross pleural thickening at the right base.

Lipiodol was injected into the pleural cavity in one patient (case 42). Subsequent radiographs failed to demonstrate a hepato-pleural communication.

PLEURAL ASPIRATION

On the basis of the findings at aspiration, the clinical presentation and the response to treatment, patients with pleural amoebiasis were divided into 2 groups.

The first of these, the presuppurative group, consisted of 5 patients (cases 31 - 35) with presuppurative effusions and a further 2 patients (cases 40 and 48) who presented with serous effusions which were subsequently replaced by suppurative effusions. From 2 patients (cases 32 and 35) serous fluid was aspirated and from 3 (cases 33, 40 and 48) serosanguineous fluid. Attempted aspiration was unsuccessful in the remaining 2 patients (cases 30 and 34); these were regarded on the basis of their clinical presentation and response to treatment to be suffering from presuppurative rather than suppurative effusions. Pus was obtained at the 2nd aspiration in 2 patients (cases 40 and 48) indicating progression to the suppurative stage. In addition to those mentioned above there were 3 patients (cases 13,14 and 22) in the pulmonary group who were shown by aspiration to have presuppurative effusions.

Examination of these serous or seroeanguineous effusions showed them to be exudates. Microscopy showed red blood cells in most with polymorphs predominating in the white cell series. No amoeba, acid-fast bacilli or pyogenic bacteria were identified or cultured.

The second of these groups, the suppurative group, consisted of 15 patients (cases 36 - 50). Pus was aspirated from the pleural cavities of 13 of these patients. One (case 38) died before aspiration could be attempted and the remaining patient (case 47) was not aspirated.

For purposes of drainage 4 of these 13 patients required a single aspiration (cases 39,43,45 and 48), 3 from 2 to 5 aspirations (cases 37,40 and 49) and 6 more than 5 aspirations (cases 36,41,42,44, 46 and 50). One patient (case 41) was aspirated 17 times.

More than 20 litres of pus was aspirated from these patients, the largest quantity obtained from a single patient was 10,950 ml. (case 41), and the largest quantity obtained at a single aspiration was 2,800 ml. (case 42).

Typical anchovy sauce pus was aspirated from 9 patients (cases 37,39,40,41,42,44,46,48 and 49), yellow pus from 3 patients (cases 36,43 and 50) and blood-stained pus from one patient (case 45). Initial aspirations in 2 patients (cases 40 and 48) in this group yielded seroeanguineous fluid whilst on subsequent aspirations

/pus ...

pus was obtained. The pleural lesions in these patients were considered to have progressed from a presuppurative to a suppurative stage.

Trophozoites of E.histolytica were identified on direct examination of the aspirate of 6 patients (cases 37,42,44,45,46 and 50). Gram-negative bacilli were cultured from the pus of one patient (case 37). Acid-fast bacilli were absent on direct examination and culture in each instance.

LIVER ASPIRATION

Aspiration of a liver abscess was attempted in 7 patients with success in 5 (cases 31,32,34,35 and 48). The largest quantity of pus removed at a single aspiration was 1,180 ml. (case 35). Two patients required second aspirations (cases 34 and 48).

Typical anchovy sauce pus was aspirated from 2 patients (cases 34 and 48), blood-stained pus from 2 patients (cases 31 and 32) and yellow pus from one patient (case 35).

Trophozoites of E.histolytica were identified in the pus from one patient (case 35). The liver pus was sterile on bacterial culture in each of the 5 patients.

SPUTUM EXAMINATION

Trophozoites of E.histolytica were found in the sputum of one (case 45) of the 9 patients with a productive cough in this group.

STOOL EXAMINATION

Trophozoites of E.histolytica were found in the stools of 3 patients (cases 31,48 and 49).

TREATMENT

Nineteen of 20 patients in this group received the tissue amoebicides emetine hydrochloride and chloroquine (sulphate or phosphate) with the intestinal amoebicide diiodohydroxyquinoline. Treatment and dosage schedules were similar to those employed in the pulmonary group. In one instance (case 35) dehydroemetine was used in place of emetine. One patient (case 38) died prior to receiving specific therapy. Thirteen patients received the tissue amoebicides emetine and chloroquine from the 1st day, 3 from the 2nd day, 2 on the 3rd day and one within the 1st week.

Antibiotics were employed in addition to specific therapy in 10 patients, the remaining 9 receiving only amoebicidal drugs.

Blood transfusions were administered to 4 patients (cases 36,37,47 and 50).

Drainage Procedures: The details of pleural and liver aspiration have been previously recorded.

The site of maximum dependancy or dullness was chosen for pleural aspirations and as much pus as possible was removed at each attempt. The procedure was repeated at 2 to 3 day intervals until

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less than 100 ml. of pus was obtained or adequate radiological resolution demonstrated.

The indications for liver aspiration were identical to those employed in the pulmonary group.

CLINICAL PROGRESS

Table 9 records the details of progress in 20 patients with pleural amoebiasis. There were 4 deaths (cases 37,38,41 and 43) and 16 recoveries. The diagnosis in 2 patients (cases 38 and 43) was only established at necropsy.

The details of 2 (cases 39 and 48) of the 16 survivors have not been included in the estimate of the average duration of hospitalisation, pyrexia or weight increase for the group as in these patients additional complications prolonged hospitalisation and pyrexia. In 14 of the 16 survivors the average duration of hospitalisation was 32 days and the average duration of pyrexia following treatment 5 days. The weight increase during hospitalisation averaged 8 lbs. per patient.

Noticeable clinical improvement was noted in the chest signs of each of the 16 survivors. Fourteen of these had persisting signs at the right base at discharge and one patient (case 40) at the left base.

Right upper quadrant guarding and tenderness cleared in each of the 14 survivors who presented with these signs, usually within the

/1st ...

TABLE 9

CLINICAL PROGRESS OF 20 PATIENTS WITH PLEURAL AMOEBIASIS

Observations		Total No. Of Patients Observed	Per Cent
Deaths	4	20	20
Average duration of pyrexia (days after treatment)	5	14	-
Average duration of hospitalisation (days)	32	14	-
Average weight increase in lbs.	8	14	-
Signs at the right lung base at discharge	14	15	93
Resolution of abdominal tenderness	14	14	100
Absence of hepatomegaly at discharge	9	11	82
Follow-up progress maintained	7	7	100

week of treatment. Of the 11 survivors who presented with a tender hepatomegaly a non tender hepatomegaly persisted in 2 (cases 31 and 39).

Seven patients (cases 31,34,36,42,47,48 and 50) attended hospital regularly for periods of one to 18 months after discharge. Six of these patients maintained their clinical progress and one (case 44) relapsed after one month.

Presuppurative Group: There was no apparent residual disability in any of the 5 recoveries (cases 31 - 35) in this group and each of these patients was able to pursue his normal occupation. There was more rapid improvement of signs in the right chest in patients in the presuppurative group (cases 31 - 35) than of those in the suppurative group (cases 35 - 50). Each of the 5 patients (cases 31 - 35) in the presuppurative group had residual signs of dullness and decreased air entry at the base of the right chest as discharge and in 2 (cases 31 and 34) the signs had cleared at one month after discharge.

Suppurative Group: Four (cases 37,38,41 and 43) of the 15 (cases 35 - 50) patients in the suppurative group died and 11 made a satisfactory recovery.

In 2 patients (cases 39 and 48) hospitalisation was prolonged because of additional complications. Of those who died, the first (case 37) was critically ill and emaciated and died on the 9th day from a chronic amoebic empyema with a bronchopleural fistula; the 2nd (case

/38)...

38) died from an amoebic brain abscess; the 3rd (case 41) responded poorly to treatment, developed a left lobe liver abscess and died in the 14th week; and the 4th (case 43) died in a state of shock on the 6th day following rupture of a liver abscess into the pleural cavity.

Eight of the 11 patients in this group who recovered had persisting signs of dullness and diminished air entry at the base of the right chest at discharge and in one patient (case 40) these signs persisted at the base of the left chest. One patient (case 44) with an encysted empyema relapsed at one month after discharge. Further courses of emetine and chloroquine were given and the effusion was drained by an intercostal tube.with a satisfactory response. In a further patient (case 42) signs in the right chest, attributed to gross pleural thickening, persisted at 18 months. Although clinical signs persisted at the base of the chest in 8 of the 11 patients who recovered from an amoebic empyema, these produced no residual disability with the exception of the patient (case 42) with gross pleural thickening who became dyspnoeic on strenuous exertion.

HAEMATOLOGICAL PROGRESS

There was an average rise of 3 g. per patient in the haemoglobin levels of the 16 survivors. Four of these patients (cases 36, 37,47 and 50) received blood transfusions.

The white cell count at discharge was 10,000 per c.mm. or less in 9 of the 11 survivors in whom it was raised on admission.

A significant decrease in the sedimentation rate was noted

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in the 7 survivors in whom serial estimations were obtained.

RADIOLOGICAL PROGRESS

Table 10 shows the details of the radiological progress in the 16 recoveries from pleural amoebiasis. Chest radiographs at discharge showed complete clearing of the effusions in 9 (cases 32,33,36,39,40,45,48,49 and 50) of the 16 survivors. Small residual effusions were noted in 7 (cases 31,34,35,42,44,46 and 47). Two (cases 31 and 34) of these residual effusions cleared within one month of discharge, 2 (cases 46 and 47) persisted at one and 4 months respectively and one (case 44) increased in size and required further drainage. Of the remaining 2, one patient (case 42) developed gross pleural thickening and one patient (case 35) failed to reattend.

Radiological changes suggestive of pleural thickening were present at discharge in 8 patients (cases 32,36,40,42,45,46,49 and 50). In 7 of these the changes were minimal. The remaining patient (case 42) had evidence of gross pleural thickening at the right base at 6 months with some resolution at one year and further resolution after 18 months. Bronchography in this patient (case 42) showed that the pleural thickening had distorted the right middle and lower lobe bronchi with partial collapse of these lobes.

Elevation of the right diaphragm persisted at discharge in 11 of the 13 patients in whom this was noted during hospitalisation.

Serial chest films were obtained in 7 patients (cases 31,

/34 ...

TABLE 10

RADIOLOGICAL PROGRESS OF 16 PATIENTS WITH PLEURAL AMEBIASIS

Observations		Total No. of Patients Observed	Per Cent
Clearing of pleural effusion	9	16	56
Small residual pleural effusions	7	16	44
Minimal pleural thickening	7	16	44
Gross pleural thickening	1	16	6
Elevated right diaphragm	11	13	85
Follow-up radiological progress maintained	6	7	86

34,42,44,46,47 and 50) for periods of one to 18 months after discharge. Radiological deterioration was seen in only one instance (case 44).

NECROPSY FINDINGS

Permission for necropsy was obtained in 4 instances (cases 37,38,41 and 43). At necropsy in each of these patients there was evidence that an amoebic liver abscess had extended through the right hemidiaphragm into the pleural cavity. In one patient (case 38) the liver abscess had healed but the track into the right pleural cavity persisted and an amoebic brain abscess was found in the left occipital region. A further patient (case 41) was found to have multiple amoebic liver abscesses with amoebic peritonitis. Three patients (cases 37, 38 and 43) had evidence of amoebic colitis.

A BRIEF REVIEW OF THE LITERATURE WITH A DISCUSSION OF THE FINDINGS IN THIS STUDY

HISTORICAL REVIEW

"My bowels are troubled,
my liver is poured upon the earth"
Lamentations, 2, 11.

It has been suggested that Galen described a patient with pulmonary amoebiasis (Blanc and Seguier, 1950) but more convincing descriptions can be found in the literature of the last century (Morehead, 1860; Kelsch and Kiener, 1884). In 1860 Rouis reported a patient with a liver abscess who recovered after coughing up large quantities of bile-stained sputum.

The beginning of our understanding in amoebiasis dates back to 1875 when Lösch, working in St.Petersburg, recognised amoebae in the stools of a patient with chronic relapsing dysentery. He gave an accurate description of the parasite which he named Amoeba coli. Whilst en route to India in 1883 Koch stopped in Egypt and observed amoebae in ulcers of the large bowel and in the walls of a liver abscess. In 1886 - 1887 Kartulis, working in Alexandria, identified amoebae in dysenteric stools, in sections of the large gut and in the pus of liver abscesses. He was amongst the first to claim this amoeba as the cause of "tropical dysentery" and liver abscess and subsequently identified the parasite in a brain abscess (Kartulis, 1904).

The classic treatise published by Councilman and LaFleur (1891) placed the pathological and clinical entities of intestinal, hepatic, and hepatopulmonary amoebiasis on a firm foundation. Included

/in ...

in their study is an unsurpassed description of the clinical and pathological findings of 4 patients with hepatopulmonary amoebiasis. Three of these were shown at necropsy to have an amoebic lung abscess communicating through the diaphragm with an amoebic liver abscess and in one there was a bronchopleural fistula connecting the lung abscess with an amoebic empyema. These workers recorded an excellent and detailed clinical description of the right basal syndrome of hepatopulmonary amoebiasis as it presented in their patients. They regarded the character of the sputa produced as pathognomonic of an abscess of the liver perforating into the lung and were the first to describe amoebae in the sputa of patients with hepatopulmonary amoebiasis.

In 1895 Bertrand and Fontan in a treatise on amoebic liver abscess, reported 16 patients who coughed up the contents of their liver abscess. Approximately half of these patients recovered, some following the spontaneous evacuation of the contents of the liver abscess through a bronchus and others following surgical drainage of the liver abscess.

Bunting in 1906 published one of the first reports of haematogenous or primary pulmonary amoebiasis. In his patient firm consolidated nodules were found in the lung at necropsy and in each of these amoeba were demonstrated at histology. He suggested that in this presentation of pulmonary amoebiasis the probable route by which the amoeba gain access to the lung is by direct embolism to the lung through

/the ...

the circulation from the colon. Tuffier in 1908 is reported to have cured a patient with a primary amoebic lung abscess by operative interference (Manson-Bahr, 1923).

A milestone in the management of hepatic and hepatopulmonary amoebiasis was reached with the introduction of emetine by Rogers in 1912. Against considerable opposition he established the basis of present day treatment when he proposed closed drainage of the liver abscess by needle aspiration coupled with the administration of emetine as the treatment of choice in this condition (Rogers, 1912). Following Roger's lead, Chauffard in 1913 cured a patient with an amoebic liver abscess and hepatobronchial fistula with emetine.

The above brief review shows that by 1900 there was a considerable knowledge of the etiology, pathogenesis, pathology and clinical presentation of hepatopulmonary amoebiasis. In the early years of this century descriptions of the entity primary pulmonary amoebiasis appeared in the literature and the use of emetine in the treatment of hepatopulmonary amoebiasis was advocated. Events which have contributed to our understanding of pleuropulmonary amoebiasis since 1920 will be mentioned in the review of the literature which follows.

INCIDENCE

Table 11 shows the reported frequency with which pleuropulmonary amoebiasis is associated with intestinal and hepatic amoebiasis. Amongst 338 patients with amoebic dysentery Ochsner and DeBakey (1935) found 7 (1.8 per cent) with pleuropulmonary amoebiasis. Radke (1951) and Rodrigues and Adrianza (1950) found that only one per cent of patients with symptomatic intestinal amoebiasis have associated pleuropulmonary amoebiasis.

Huard and Meyer-May (1936) and Ochsner and DeBakey (1936) reported that pleuropulmonary complications occurred in 15.7 per cent of their patients with amoebic liver abscesses. In their remarkable review of 2,490 cases of amoebic liver abscess collected from the literature Ochsner and DeBakey (1936) found 407 (15.8 per cent) thoracic lesions; 209 (8.3 per cent) of these were pulmonary complications and 198 (7.5 per cent) were pleural complications. More recently Kean et al. (1956) in a pathological study of 90 amoebic liver abscesses found pleuropulmonary complications in 24.4 per cent and Lamont and Pooler (1958), in a clinical study of 250 patients with amoebic liver abscesses, reported intrathoracic extension in 10.4 per cent.

The findings quoted above show that amoebic liver abscess is far more commonly associated with pleuropulmonary amoebiasis than is symptomatic intestinal amoebiasis and this has certainly been my experience of this condition at this hospital. I have mentioned previously that the present study lasted 3 years and that by no means

/all ...

TABLE 11

THE FREQUENCY WITH WHICH PLEUROPULMONARY AMOEBIASIS IS ASSOCIATED WITH INTESTINAL AND HEPATIC AMOEBIASIS

(a) Intestinal Amoebiasis

Author	Symptomatic Intestinal Amoebiasis	Pleuro-pulmonary lesions	Per Cent
Ochsner and DeBakey (1935)	338	7	1.8
Radke (1951)	101	1	1.0
Rodrigues and Adrianza (1950)	1000	10	1.0

(b) Hepatic Amoebiasis

Author	Amoebic Liver Abscesses	Pleuro-pulmonary lesions	Per Cent
Huard and Meyer-May (1936)	150	21	15.7
Ochsner and DeBakey (1936)	95	15	15.7
Kean et al. (1956) (pathological study)	90	22	24.4
Lamont and Pooler (1958)	250	26	10.4

all patients with pleuropulmonary amoebiasis admitted to this hospital during this period have been included. It was, therefore, impossible from my material to determine either the frequency with which pleuropulmonary amoebiasis is associated with amoebic dysentery or amoebic liver abscess at this hospital.

When discussing the frequency with which pleuropulmonary lesions complicate an amoebic liver abscess it should be stated whether one is referring to only those lesions which have resulted from frank rupture or extension of the liver abscess through the diaphragm or to all pleuropulmonary lesions associated with the underlying liver abscess. This point is illustrated by the figure of 10.4 per cent quoted from Lamont and Pooler's study (1958) which represents only those abscesses which extended through the diaphragm and would have been considerably higher had all thoracic lesions associated with an underlying liver abscess been included.

AGE

Figure 5 shows the age distribution in this series as compared with the findings of Ochsner and DeBakey (1936). It can be seen that the highest incidence was in the 3rd, 4th and 5th decades in both series. This distribution is similar to that reported in amoebic liver abscess by Klatskin (1946) and De Silva (1946).

In recent years Salas, Scragg and others have reported the occurrence of pleuropulmonary amoebiasis in infants (Salas, 1958;

/Scragg ...

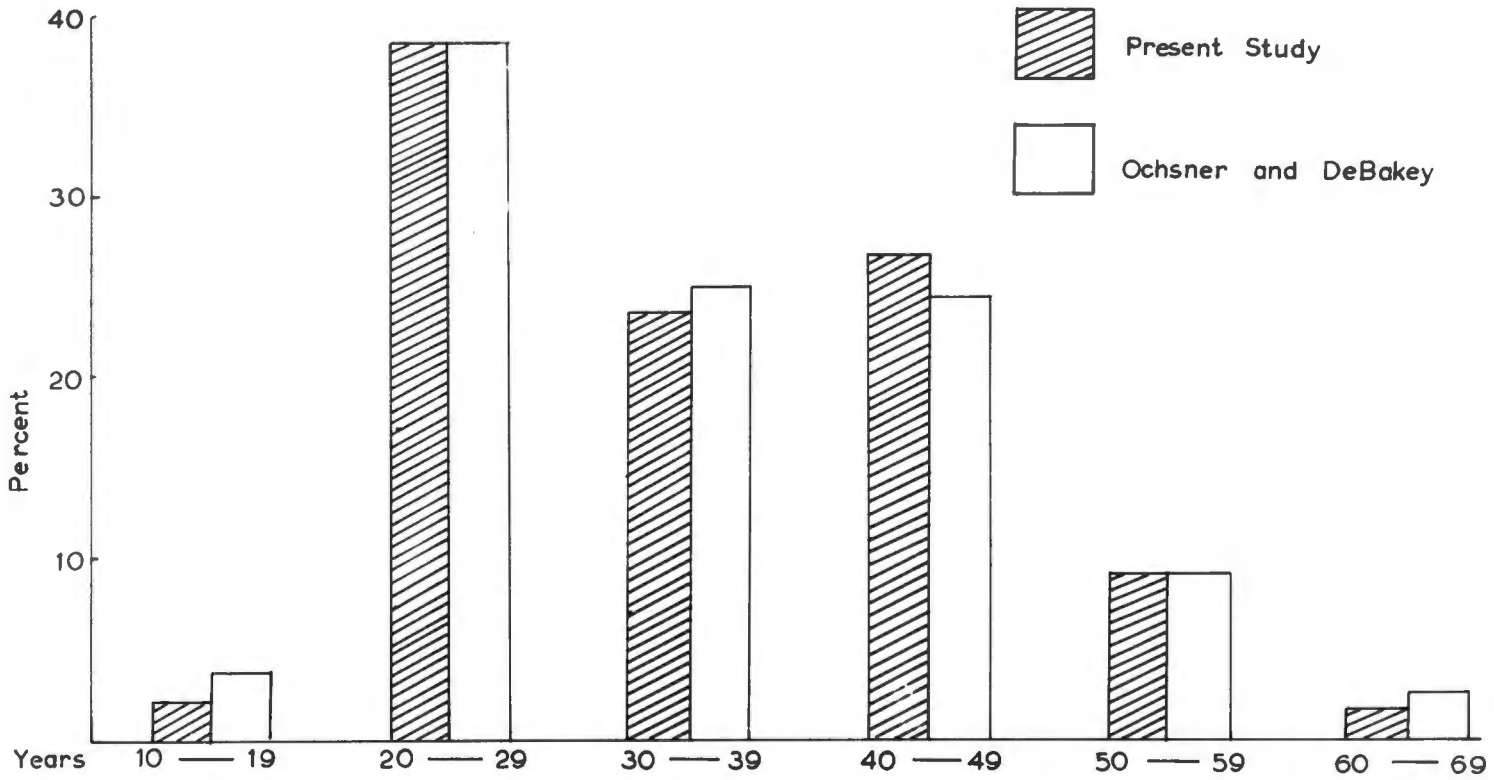

FIG.5. AGE DISTRIBUTION IN PLEUROPULMONARY AMOEBIASIS.

Finding in this study compared with those of Ochsner and De Bakey (1936)

Seragg, 1960; Jayaratne and De Silva, 1960; Macdougall, 1960). Although infants are seen with pleuropulmonary amoebiasis at this hospital (Seragg, 1960), no examples were included in this study.

SEX

Takaro and Bond (1958) stated that the sex incidence of pleuropulmonary amoebiasis parallels that of hepatic amoebiasis. Nine per cent of their collected series were females. No females were included in this study but in my experience the sex distribution of pleuropulmonary amoebiasis at this hospital is similar to that quoted above.

PATHOGENESIS

THE PRECURSOR OF THE PLEUROPULMONARY LESION

From the site of the primary infection in the large bowel the amoeba may invade nearly any organ in the body (Freedman and Cleve, 1952). The commonest situation of these extra intestinal lesions is the liver followed by the lungs and pleura (Ochsner and DeBakey, 1936). Spread of the amoeba to the liver is considered to occur via the portal venous system, whilst in the liver invasion of the parenchyma with multiplication of the amoeba, results in tissue necrosis and abscess formation (Rogers, 1922; Palmer, 1938).

Bockless (1950) favoured amoebic hepatitis rather than amoebic liver abscess as the origin of the pulmonary lesions in his patients. Carruthers (1947) stated that plastic pleurisy may result from an underlying hepatitis. Other workers have postulated that with spread of E.histolytica from the bowel the vascular network of the liver acts as a primary filter and the pulmonary capillary bed as a secondary filter (Blanc and Siguier, 1946).

Plausible as the above postulates may seem, they may be seriously questioned. Although pathological descriptions are available of wide spread invasion of the liver with E.histolytica in the absence of suppuration (Chatzidakis, 1953; Dexiades et al., 1961), these are rare, and in some instances have had their validity questioned (Adams, 1961). The existence of a pathological basis for the frequently diagnosed

/clinical ...

clinical entity amoebic hepatitis has been disputed. Kean (1955), Lamont and Pooler (1958), and numerous other workers have been unsuccessful in attempts to demonstrate pathological evidence for this clinical entity (Da Silva, 1950; Chaudhuri and Saha, 1956; Powell et al., 1959).

The experience of most workers is that an amoebic liver abscess is the usual precursor of the thoracic lesion (Miginiac, 1922; Vergos and Hermanjat-Gerin, 1932; Ochsner and DeBakey, 1936; Lamont and Pooler, 1958; Wilmot, 1962). Necropsy and clinical findings in this study support this view. The entity of diffuse amoebic hepatitis without suppuration has not been seen in the pathology department of this hospital, which has an extensive experience of hepatic amoebiasis (Wainwright, 1962). In my patients an amoebic liver abscess as opposed to amoebic hepatitis, was the considered precursor of the thoracic lesion in each instance.

PATHOGENIC PATHWAYS TO THE LUNGS AND PLEURA (See Figure 6)

Direct Extension: The majority of pleuropulmonary amoebic lesions are situated at the right lung base (Ochsner and DeBakey, 1936; Takara and Bond, 1958). Necropsy evidence and clinical presentations indicate that rupture or direct extension of an amoebic liver abscess through the right diaphragm is the usual pathogenic basis for these thoracic lesions (Vergos and Hermanjat-Gerin, 1932; Huard and Meyer-May, 1936; Ochsner and DeBakey, 1936; Lamont and Pooler, 1958). Akenhead (1948) considered that more than 75 per cent of pleuropulmonary

/amoebic ...

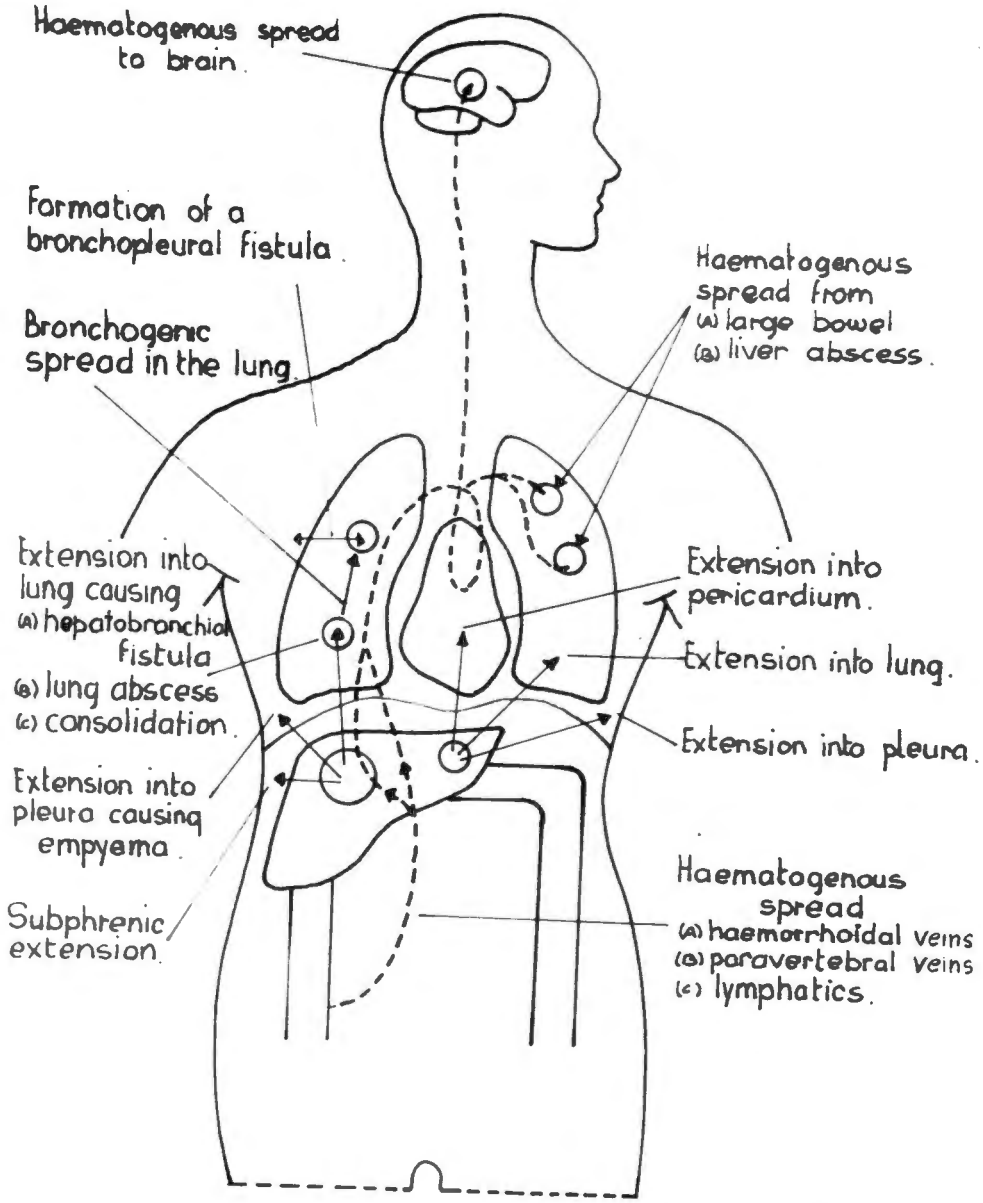

Fig. 6. Postulated pathogenic pathways in thoracic amoebiasis.

amoebic lesions result from direct extension of an underlying amoebic liver abscess.

The frequency of direct extension of an amoebic liver abscess through the diaphragm in the pathogenesis of thoracic amoebiasis is doubtlessly dependent on the situation of the liver abscess. Most studies have shown that amoebic abscesses have a predilection for the right lobe of the liver with a frequency which ranges from 70 to 90 per cent (Vergne and Hermenjat-Gerin, 1932; Huard and Meyer-May, 1936; Ochsner and DeBakey, 1939; Lament and Peeler, 1958). Furthermore, these abscesses are commonly situated in the upper aspect of the right lobe of the liver immediately beneath the right diaphragm (Councilman and LaFleur, 1891; Ochsner and DeBakey, 1936).

Amoebic pleural and pulmonary lesions are rarely situated in the left chest (Wilmot, 1962). Ochsner and DeBakey (1936) made no mention in their review of lesions in this situation, whilst recently Takaro and Bond (1958) reported left sided lesions in 10 per cent of their collected series. To date Lament and Peeler (1958) have reported the largest number of left basal lesions in a personal series. Of their 250 amoebic liver abscesses 26 ruptured through the diaphragm and 5 (19 per cent) of these extended into the left chest. Other workers who have reported left basal pleural or pulmonary lesions have been Dansde and Lavignolle, 1947; Chakravarti, 1951; Ghosh, 1954; Langeron et al., 1958; Alkan et al., 1961).

Postulates such as the smaller size of the left lobe of the liver (Shaw, 1949; Talbot, 1951), the division of the portal blood flow

/into ...

into two streams (Serego, 1901), and the greater width and straighter course of the right portal vein (Elsberg, 1906) have been proffered to explain the relatively infrequent occurrence of amoebic abscesses in the left lobe of the liver. Whatever the explanation, this lesser tendency coupled with the fact that anatomically the left lobe of the liver has a limited relationship to the left lung base, suggests the reason why extension of a left lobe liver abscess into the left lung or pleural space is an uncommon occurrence.

In a fairly extensive experience of thoracic amoebiasis at this hospital I have found direct extension of a liver abscess through the diaphragm to be responsible for almost all suppurative amoebic pleuropulmonary lesions. Clinical and necropsy findings in this study support this statement. In 47 patients there was evidence of extension through the right diaphragm and in 3 (cases 6,40 and 48) evidence of extension through the left diaphragm. These findings are of obvious importance to the clinician and their clinical implications will be discussed in due course.

Necropsy findings show that in the process of trans-diaphragmatic extension the superior surface of the liver, the overlying diaphragm and the lung base are bound together by adhesions (Rogers, 1922; Verges and Normenjat-Gerin, 1932; Huard and Meyer-May, 1936; Lamont and Pooler, 1938). These adhesions result from the inflammatory reaction at the periphery of the extending liver abscess. It has been suggested that if the potential pleural space is obliterated by these

/adhesions ...

adhesions extension or rupture occurs into the lung base. When, however, this space is not obliterated rupture into the pleural cavity takes place with a resultant amoebic empyema (Ochsner and DeBakey, 1936). In some instances prior to extension through the diaphragm rupture into the subphrenic space may occur with the formation of a subphrenic abscess (Huard and Meyer-May, 1936; Foote, 1947; Kossalka et al., 1949; Peak and Eskridge, 1950).

Necropsy findings in this study support the above statements (cases 3,6,37,38,41 and 43). Dense adhesions binding the superior surface of the liver, the diaphragm, and the lung base were found. In most there was a communication or fistula linking the liver abscess with either the pleural space or the base of the lung. In one instance (case 3) the communication consisted of a large hepatopulmonary abscess with half its substance in the liver and the remainder in the lung. In another patient (case 6) the hepatopulmonary fistula had healed but a pulmonary gastric fistula connecting a left basal amoebic lung abscess with the stomach was demonstrated.

Diaphragmatic fistula or communications in pleuropulmonary amoebiasis may vary in size from one to several centimeters and occasionally multiple tracks may be found (Huard and Meyer-May, 1936).

There have been reports of suppurative basal pleuropulmonary lesions with an underlying liver abscess in which the diaphragm at autopsy was found to be intact. It has been postulated in such instances that spread through diaphragmatic lymphatics (Leisen, 1908; Sambuc, 1913;

/Beckless ...

Bockless, 1950), or through the trans-diaphragmatic capillaries (Rodriguez and Adriansza, 1950) was the likely mode of extension. There were no examples of this type of suppurative pleuropulmonary amoebiasis seen at necropsy in this study.

Haematogenous Spread: Embolic or haematogenous spread to the lungs may originate from the primary amoebic focus in the bowel or the secondary amoebic focus in the liver (Deaton and Garrett, 1956). Features suggesting a haematogenous origin are, the occurrence of an amoebic lung abscess in a situation remote from the diaphragm (Ochsner and DeBakey, 1936) and the occurrence of multiple amoebic lung abscesses (Chatterjee and Sen Gupta, 1949). Pulmonary amoebic lesions of haematogenous origin are rare (Hanson-Behr, 1923) and the information available has been derived mainly from isolated case reports (Bunting, 1906; Simmain, 1914; Hanson-Behr, 1923; Girgis, 1939; Verner, 1948; Chatterjee and Sen Gupta, 1949; Ginsberg and Miller, 1952; Deaton and Garrett, 1956).

Haematogenous spread from a liver abscess: The literature contains a number of descriptions of amoebic liver abscesses with associated amoebic lung abscesses in situations remote from the diaphragm (Harrington, 1930; Girgis, 1939; Lindsay et al., 1951; Cresnier and Darbon, 1952). As mentioned above, Ochsner and DeBakey (1936) regarded the occurrence of an amoebic lung abscess in a situation remote from the diaphragm as evidence of haematogenous spread. Their classification

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can be seen in table 12. In their review of 158 patients with pulmonary amoebiasis these workers considered 16 to have had haematogenous pulmonary amoebiasis and an associated independent amoebic liver abscess.

Girgis (1939) reported 2 patients in whom at necropsy there were multiple amoebic lung abscesses and amoebic liver abscesses with no evidence of diaphragmatic involvement. In one patient he was able to demonstrate emboli in the pulmonary artery which contained trophozoites of E.histolytica. Although both patients had amoebic ulceration of the bowel, embolic spread to the lungs from the liver abscess, rather than the intestinal lesion, was favoured.

Haematogenous spread from the large bowel: Postulated vascular pathways by which the amoeba may reach the systemic circulation and thence the lungs are as follows:

From the large bowel via the portal vein to the liver and thence to the hepatic vein and inferior vena cava (Petsetakis, 1931).

Via the haemorrhoidal veins to the systemic veins (Petsetakis, 1931; Harrington, 1930; Deaton and Garrett, 1956).

Via the bowel lymphatics and thoracic duct to the left subclavian vein (Petsetakis, 1931; Barbiera, 1939).

Via the paravertebral veins to the systemic circulation (Sullivan and Bailey, 1951).

In their review of 158 patients with pulmonary amoebiasis

/(see ...

(see table 12) Ochsner and DeBakey (1936) considered 22 to have had haematogenous pulmonary amoebiasis without associated liver involvement, a finding suggesting that spread had occurred from the primary lesion in the bowel. Others who have reported pulmonary amoebiasis in the absence of signs and symptoms suggestive of hepatic amoebiasis have been Hameed (1945), Chaudhuri and Chaudhuri (1946), Bilimagady (1947) and Sullivan and Bailey (1951).

In the absence of evidence of hepatic amoebiasis the term "primary pulmonary amoebiasis" has been applied to amoebic lung pathology considered to have originated from haematogenous spread from the bowel (Manson-Bahr, 1923; Dorner and Friedlander, 1941; Hameed, 1945; Mukherjee, 1949; Deaton and Garrett, 1956). This terminology has been criticised as confusing on the grounds that pulmonary amoebiasis is never primary but always secondary to either intestinal or hepatic amoebiasis (Takaro and Bond, 1958; Wilmot, 1962).

There were few patients in this study in whom haematogenous pathways could be proposed with any confidence as the source of thoracic lesions. Undoubted evidence of haematogenous spread was found in 2 patients (cases 38 and 58) in whom amoebic brain abscesses were demonstrated at necropsy. (The associated pleural lesion in one of these patients (case 38) was shown to have resulted from direct extension of a liver abscess through the diaphragm). A further 3 patients (cases 2,3 and 13) presented with radiological evidence of

/lesions ...

TABLE 12

THE INCIDENCE OF HAEMATOGENOUS PULMONARY AMOEBIASIS

(Ochsner and DeBakey, 1936)

	No. of Cases	Per Cent
Haematogenous Pulmonary Amoebiasis without liver involvement	22	14
Haematogenous Pulmonary Amoebiasis and independent liver abscess	16	10
Pulmonary abscess extending from a liver abscess	58	38
Bronchohepatic fistula with little pulmonary involvement	30	20
Empyema extending from amoebic liver abscess	27	18
	153	100

lesions in situations remote from the diaphragm, and in one of these (case 3) multiple left upper lobe lung abscesses were found at necropsy. Although haematogenous spread to the lungs may have occurred in these 3 patients, each of them had associated evidence of right basal pulmonary involvement, and the usual pathway of direct extension through the diaphragm followed by subsequent bronchogenic spread seemed a more likely sequence.

Pulmonary amoebiasis of haematogenous origin is rare. Because of this its occurrence is more likely to prompt a report in the literature than the common variety following direct extension of a liver abscess. This is the probable explanation of the disproportionately large numbers of this type of pulmonary amoebiasis included in the reviews by Ochsner and DeBakey (1936) and Takaro and Bond (1958) which, judging from my experience, give a false impression of its frequency.

Inhalation of the Amoeba: There have been those who have suggested that inhalation of the amoeba may be responsible for the doubtful clinical entity of amoebic bronchitis (Petsetakis, 1923; Panayatalou and Netter, 1924; Netta, 1938). The term, "primary pulmonary amoebiasis", could be more correctly applied to pulmonary amoebiasis of such unlikely origin. To date, there has been insufficient evidence to consider inhalation of the amoeba as a possibility in the pathogenesis of pulmonary amoebiasis.

CONCLUSIONS

Judging from reports in the literature pleuropulmonary amoebiasis may be expected to occur in less than 2 per cent of patients with symptomatic intestinal amoebiasis and in 10 to 20 per cent of those with hepatic amoebiasis. The highest incidence is found in males in the 3rd and 4th decades. In females and children the condition is rare.

Findings in this study suggested that an amoebic liver abscess as opposed to the doubtful clinical entity of diffuse amoebic hepatitis was the precursor of the thoracic lesions.

Clinical, radiological and necropsy findings confirmed the view that the large majority of pleuropulmonary amoebic lesions result from direct extension of an amoebic liver abscess through the diaphragm, commonly into the right chest and uncommonly into the left chest.

Haematogenous pulmonary amoebiasis is rare and may result via vascular spread from the primary amoebic focus in the large bowel or from the secondary amoebic focus in the liver. There were few instances in this study in which haematogenous pathways could be implicated in the pathogenesis. It is suggested that the frequency of this type of pulmonary amoebiasis as reported in reviews of the literature is erroneously high.

There is insufficient evidence to consider inhalation of the amoeba as a possibility in the pathogenesis of pleuropulmonary amoebiasis.

CLINICAL FINDINGS IN PLEUROPULMONARY AMOEBIASIS

Depending on the clinical presentation, patients with pleuropulmonary amoebiasis may be divided into two groups: (i) Those who present with the clinical feature of associated hepatic amoebiasis, a presentation described in the literature under such headings as "Secondary Pulmonary Amoebiasis" (Dorner and Friedlander, 1941), "Hepato-pulmonary Amoebiasis" (Singh and Jolly, 1962), and "Thoracohepatic Amoebiasis" (Koszalka et al., 1949). (ii) Those in whom there is no evidence of liver involvement, a presentation described in the literature under headings such as "Primary Pulmonary Amoebiasis" (Manson-Bahr, 1923; Deaton and Garrett, 1956) or "Hematogenous Pulmonary Amebiasis without Liver Involvement" (Ochsner and DeBakey, 1936).

GROUP 1 - HEPATOTHORACIC AMOEBIASIS

On the basis of clinical and radiological findings, and the nature of the pleural fluid, hepatothoracic amoebiasis has been documented in the literature under two headings. The first of these is the commonly reported suppurative presentation, and the second the rarely reported non-suppurative or presuppurative presentation.

The large majority of suppurative thoracic amoebic lesions result from direct extension of an amoebic liver abscess through the diaphragm and this was the probable origin of all such lesions in this

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study. There follows a brief review of the literature and discussion of the clinical presentation in this, the common, variety of pleuropulmonary amoebiasis.

Suppurative Hepatothoracic Amoebiasis

Duration of Symptoms: The experience of previous workers and the findings in this study indicate that the duration of symptoms in thoracic amoebiasis is usually measured in weeks or months (Chakravarti, 1951,1952,1953; Abdel-Hakim and Higazi, 1958). Occasionally the onset may be acute as in Manson-Bahr's description of a "Hong Kong" liver abscess suddenly coughed up on a British golf course (Manson-Bahr, 1944). Sometimes symptoms may exist for years before their significance is appreciated (Abdel-Hakim and Higazi, 1958).

Past History: Physicians in temperate climates have emphasized the importance of a history of exposure or residence in endemic areas (Wright, 1941; Cenan et al., 1950; Sullivan and Bailey, 1951). Patients may admit to a previous history of diarrhoea or dysentery (Ochsner and DeBakey, 1936; Abdel-Hakim and Higazi, 1958) but in the majority such a history is not obtained (Alareen, 1942,1954; Chakravarti, 1951; Singh and Jelly, 1962).

It has been my experience that the majority of patients with pleuropulmonary amoebiasis do not admit to past or present symptoms of intestinal amoebiasis. Only 26 per cent of patients in this study admitted to a previous episode of dysentery whilst 12 per cent

/experienced ...

experienced dysenteric symptoms on admission.

Constitutional Symptoms and Signs: Fever, night sweats, weakness, weight loss and malaise may be experienced in pleuropulmonary amoebiasis (Mansen-Bahr, 1923; Ochsner and DeBakey, 1936; Coirault et al., 1955; Abdel-Hakim and Higazi, 1958).

A moderate pyrexia without conformity to a regular pattern is usual (Ochsner and DeBakey, 1936; Chakravarti, 1951) and was a feature in most of my patients, although 12 per cent remained apyrexial throughout their illness. Exceptionally patients presented with a temperature of 102 to 104°F. Such levels were usually short-lived and settled soon after commencing treatment. A persistent swinging temperature in pleuropulmonary amoebiasis may be indicative of secondary bacterial invasion (Ochsner and DeBakey, 1936).

Pale mucosae denoting clinical anaemia have been described as a frequent finding in patients with amoebic liver abscesses at this hospital (Lamont and Peeler, 1958), but there has been little mention in the literature of this finding in pleuropulmonary amoebiasis. As no less than 64 per cent of my patients had clinical signs of anaemia, it is apparent that there should be a greater awareness of this occurrence in pleuropulmonary amoebiasis.

Wasting or clinical evidence of weight loss may result from an amoebic liver abscess (Mansen-Bahr, 1931), and has been included amongst the constitutional signs encountered in pleuropulmonary

/amoebiasis ...

amoebiasis (Coirault et al., 1955; Kilgore, 1951). In chronic presentations frank cachexia may develop (Dansac and Lavignolle, 1947; Hughes and Westphal, 1947). Wasting as judged from loss of subcutaneous tissue and muscle bulk was present in 40 per cent of my patients and some were emaciated. Recent weight loss was more frequently evident and was more severe in my patients with pleural amoebiasis than in those with pulmonary amoebiasis and appeared to be related to the extent of the suppurative lesion as well as the duration of the illness.

Recently in a series of patients with bronchopulmonary amoebiasis Abdel-Hakim and Higazi (1958) reported that clubbing of the fingers was present in 61 per cent. Clubbing of the fingers was seen in approximately one-third of my patients with pulmonary amoebiasis, most frequently in those with radiological evidence of an amoebic lung abscess.

Local Symptoms: The clinical features in hepatothoracic amoebiasis resulting from direct extension include those produced by the liver abscess and those resulting from the thoracic lesion (Kilgore, 1951). Similar descriptions of the respiratory and abdominal findings in this variety of pleuropulmonary amoebiasis have been documented by the following, Ochsner and DeBakey (1936), Boekless (1950), Chakravarti (1951,1952,1953), Lamont and Pooler (1958), Takaro and Bond (1958), Abdel-Hakim and Higazi (1958) and Wilmot (1962).

The most constant symptom in hepatothoracic amoebiasis is pain situated over the right lower chest and in the right upper quadrant of the abdomen. The pain is usually continuous in character, of variable severity, may be aggravated by jarring and have features indicative of pleural involvement. Radiation may occur to the right shoulder or supraclavicular region. Less commonly with a left lobe abscess the pain may be situated in the epigastrium, involve the left lower chest and radiate to the left shoulder. The pain is nearly always associated with a cough which in the early stages may be non-productive or productive of small quantities of clear or blood-streaked sputum.

A sequence sometimes described is the onset of symptoms suggestive of an amoebic liver abscess, followed after a variable period by the sudden coughing of large quantities of reddish-brown or blood-streaked pus. This frightening event results from rupture of the liver abscess into a major bronchus. Rarely it may be the presenting feature of a clinically silent or asymptomatic liver abscess (Hansen-Behr, 1944). In the majority the presentation is not so dramatic and the complaints are of cough with a gradually increasing production of reddish-brown or blood-stained pus.

Dyspnoea has been noted as a symptom particularly following intra-pleural rupture of an amoebic liver abscess. This may be severe, accompanied by peripheral vascular collapse and followed by death (Mick, 1955).

The symptoms experienced by my patients were similar in most

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respects to those mentioned above. With few exceptions they focussed attention on the right lower chest and in many instances there was little to suggest an associated subphrenic lesion. Pain situated in the right lower chest was the most frequent symptom. The character of the pain almost always suggested pleural involvement and in some it was accompanied by a less severe, continuous pain situated in the right upper quadrant of the abdomen. In 40 per cent of my patients the pain radiated to the right shoulder tip or supraclavicular region, a symptom which was regarded as evidence of diaphragmatic involvement. In the African patient attending this hospital pain referred to the right shoulder-tip is most commonly produced by amoebic liver abscess.

The pain was invariably associated with increased coughing and no less than 76 per cent of patients complained of coughing up quantities of reddish-brown pus, blood-stained pus or pure blood. A number of patients experienced a frightening event in which, without prior warning, large quantities of pus were suddenly coughed up. In most instances, however, the onset of the productive cough was not dramatic. A cough productive of the above types of sputum was experienced by 50 per cent of patients with pleural amoebiasis and indicated that there was concomitant pulmonary involvement in these patients.

More patients in the pleural than in the pulmonary group were dyspnoeic. With large effusions this was often a distressing symptom and was sometimes accompanied by signs of shock. With the exception of this symptom there was little difference in the symptomatology of the two groups.

The Sputum: Councilman and LaFleur (1891) described the sputa coughed up by their patients as being non-offensive purifern matter of a dull brick-red, reddish-brown or rusty-brown colour. They noted that it was usually mixed with variable quantities of blood and was sometimes bile stained. They were impressed by the quantities coughed up, which varied from 25 ml. to 500 ml. When an amoebic abscess ruptures into the lung, the contents may be suddenly coughed up in mouthfuls of frothy blood and pus, but usually this process is much more gradual and small quantities are coughed up; in favourable cases the amount of expectoration gradually diminishes (Manson-Bahr, 1945). In some of the case reports reviewed by Miginiac (1922), quantities of up to 500 ml. of pus were produced daily.

Numerous adjectives with a gastronomic flavour have been employed in the description of the sputum in hepatothoracic amoebiasis, e.g. "apple butter or strawberry jelly sputum" (Webster, 1960), "catsup-like sputum" (Langston and Fox, 1947), "currant jelly and raspberry sauce sputum" (Takaro and Bond, 1958). The most frequently employed terms in the literature are "chocolate sauce or chocolate-coloured sputum" (Miginiac, 1922; Manson-Bahr, 1923; Ochsner and DeBakey, 1936) and "anchovy sauce sputum" (Dorner and Friedlander, 1941; Berne, 1942; Singh and Jolly, 1962).

Contrary to the opinion expressed by Dorner and Friedlander (1941) the sputum in hepatothoracic amoebiasis need by no means always

/be ...

be of an anchovy sauce colour. Recently Takaro and Bond (1958) stated that blood-stained pus or sputum which is in no way characteristic may be coughed in suppurative pulmonary amoebiasis following extension of a liver abscess into the lung.

Rarely the pus coughed following the extension of a liver abscess may have a bitter taste and a greenish tinge or greenish-yellow colour resulting from the presence of bile. This type of sputum is pathognomonic of a bronchobiliary fistula, examples of which have been described by Hughes and Westphal (1947), Cleve and Correa (1958) and Webster (1956,1960). Bile-stained sputum was noted in 8 of the 10 patients with pulmonary amoebiasis reported by Webster (1960).

Anchovy sauce sputa suggestive of an amoebic origin was seen in less than half my patients with a productive cough. The remainder coughed up quantities of pus which had no suggestive features. In most instances this pus contained quantities of blood and in some patients there was frank haemoptysis (case 6). There were no patients in this study who coughed up bile-stained sputum. In my experience a bronchobiliary fistula rarely develops following extension of an amoebic liver abscess into the lung. During the period of this study only 2 patients were seen with a bronchobiliary fistula at this hospital.

A noteworthy finding was the volume of pus produced. This was certainly larger than the quantities usually coughed up with a pyogenic abscess or other suppurative pulmonary conditions. It frequently

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appeared disproportionately large when compared to the extent of radiological changes in the lung, an observation also reported by Applebaum and Frankel (1948).

Local Signs: In patients with pleuropulmonary amoebiasis clinical signs are commonly found at the base of the right lung. Ochsner and DeBakey (1936) noted that with intra-pulmonary rupture the signs are usually those of consolidation or cavitation, but when rupture into the pleura occurs the clinical manifestations of a pleural effusion are found. Others have reported right basal findings suggestive of lung abscess, pneumonia, bronchopneumonia, bronchitis, dry pleurisy or a pleural effusion (Beckless, 1950; Chakravarti, 1951,1952,1953; Alarcon, 1954; Abdel-Hakim and Higazi, 1958).

On examination of the abdomen in the majority of patients the liver is enlarged and tender (Ochsner and DeBakey, 1936; Takaro and Bond, 1958; Abdel-Hakim and Higazi, 1958). Ochsner and DeBakey (1936) noted that the finding of concomitant involvement of the liver and lung is suggestive of amoebic pulmonary infection. In some patients, however, the abdominal signs of a liver abscess may be absent and as a result the amoebic etiology is not suspected (Coirault et al., 1955).

Occasionally patients may present with the abdominal signs of an amoebic liver abscess and signs of pulmonary pathology or suppuration in a situation remote from the right base or diaphragm.

Ochsner and DeBakey (1936) collected 16 such case reports from the literature and included them in their series under the heading, "Hematogenous pulmonary abscess and independent liver abscess". This presentation differs from the classical hepatothoracic presentation in that the pulmonary signs may be located in situations other than the right base. Others who have reported this variety of pulmonary amoebiasis have been Harrington (1930), Girgis (1939), Verner (1948) and Chakravarti (1951,1952,1953).

A small percentage of patients with hepatothoracic amoebiasis present with symptoms and signs in the left lower chest and epigastrium or left upper quadrant. These clinical features result from extension of a left lobe liver abscess through the left diaphragm, a sequence of events reported by Gosh (1954), Lamont and Pooler (1956), Alkan et al. (1961) and others.

Clinical signs of pulmonary or pleural involvement were nearly always confined to the right chest in my patients. Exceptions were one patient (case 6) who presented with signs at the base of the left lung and 2 (cases 40 and 48) who subsequently developed signs in this situation. A further patient (case 3) had signs in the left upper chest in addition to those at the base of the right lung. In patients with pulmonary amoebiasis and those with small to moderate sized effusions the signs were confined to the base of the right lung. Signs suggestive of an elevated right diaphragm were often more prominent in the pulmonary group than those suggestive of parenchymal involvement. It was usually impossible in these patients to determine the nature of the pulmonary

/lesion ...

lesion from the clinical signs elicited at the base of the right lung. This observation was previously reported by Osburn (1942). In my patients with pleural amoebiasis difficulty was frequently experienced in distinguishing between a small to moderate sized effusion and an elevated right diaphragm.

Some of the patients in this study with pulmonary amoebiasis had signs of a pleural effusion and a number of those with pleural amoebiasis coughed up quantities of pus. In my experience combined pleural and pulmonary lesions are a fairly common occurrence in thoracic amoebiasis. Other workers who have reported concomitant pleural and pulmonary lesions have been Hughes and Westphal (1947), Ruggieri and Cali (1951) and Takaro and Bond (1958).

Whilst the symptoms in my patients frequently focussed attention on the chest the signs elicited pointed to subphrenic as well as thoracic involvement. These included such findings as guarding and tenderness in the right upper quadrant, a tender hepatomegaly, localised intercostal tenderness and occasionally a localised or generalised swelling in the right upper quadrant of the abdomen. It was mentioned in the review of the literature that the majority of patients with pleuropulmonary amoebiasis have a tender enlarged liver. In this study this majority was small as in 40 per cent of patients the liver was not palpable. In some instances guarding and tenderness in the right upper quadrant of the abdomen interfered with palpation.

There was only one patient (case 3) in this study who could have been included in the "haematogenous pulmonary abscess and independent liver abscess" category proposed by Ochsner and DeBakey (1936). This patient had signs of pulmonary involvement in the left upper chest in addition to those at the base of the right lung. It has been my experience that clinical signs are seldom found in situations remote from the diaphragm in pulmonary amoebiasis and that the above presentation is rare.

Presuppurative Hepatothoracic Amoebiasis

The terms presuppurative or non-suppurative pulmonary amoebiasis have been applied to presentations in which there is evidence of hepatic amoebiasis with clinical and radiological pulmonary changes in the absence of a cough productive of pus (Amouille, 1941; Blanc and Siguier, 1946; Coirault et al., 1955). Coirault et al. (1955) on the basis of clinical findings and radiological changes proposed a detailed classification for this variety of pulmonary amoebiasis. In my opinion their classification is confusing and too detailed to be of practical value. They rightly stressed, however, the necessity of not missing or misinterpreting the signs of the hepatic lesion and as a result failing to appreciate the amoebic origin of the pulmonary changes. They also noted that the non-suppurative pulmonary lesion may progress to the suppurative state.

Blanc and Siguier (1946) are of the opinion that these presuppurative lesions are of haematogenous origin. This may be so

/in ...

in the uncommon presentation in which they are situated in areas of the lung remote from the diaphragm. When, however, they are situated in the right lower lung, as is usual, contiguous spread from a sub-diaphragmatic source is more likely.

Pleural effusions of a presuppurative or non-suppurative nature have been reported by Anagnostopoulos (1940), Carruthers (1947) and Ceirault et al. (1955). Patients with these lesions may present with signs and symptoms of an amoebic liver abscess and a pleural friction rub or pleural effusion at the right base. Needle aspiration is required to establish the presuppurative nature of the effusion. As with the presuppurative pulmonary lesions these effusions may progress to the suppurative state (Ceirault et al., 1955).

Clinical examples of presuppurative pulmonary amoebiasis were not included in this study. Patients are frequently seen at this hospital who present with the clinical features of an amoebic liver abscess and radiological changes at the right lung base. Such presentations, it may be argued, could reasonably be regarded as pre-suppurative lesions. In the large majority, however, the clinical features of the liver abscess dominate the presentation and there seems little point in adding the additional label of presuppurative pulmonary amoebiasis. In many of these patients the radiological changes at the right base can be ascribed to the mechanical effects of an elevated right diaphragm.

The clinical details of patients (cases 31 - 35) with presuppurative pleural lesions in this study have been recorded. In every instance these effusions were considered to have resulted from the inflammatory reaction at the periphery of an adjacent liver abscess, or adjacent pulmonary suppuration. Pleural aspiration was required to distinguish these effusions from suppurative amoebic effusions. When clinically detectable these effusions were usually small to moderate in size and, compared to the suppurative effusions, produced little in the way of local or general debility. One patient with a non-suppurative effusion subsequently developed an amoebic empyema (case 48).

GROUP XI - PRIMARY PULMONARY AMOEBIASIS (PULMONARY AMOEBIASIS WITHOUT LIVER INVOLVEMENT)

Suppurative Lesions: In 1923 Manson-Bahr reviewed the literature and reported 3 patients with pulmonary amoebiasis in whom there was no evidence of liver involvement. He considered that spread had occurred from the primary focus in the bowel in these patients and because of this referred to the presentation as "Primary Pulmonary Amoebiasis". He noted that the diagnosis was arrived at by a process of exclusion, a previous history of intestinal amoebiasis and the lasting response to emetine and ipecacuanha.

Ochsner and DeBakey (1936) included 22 reports of this variety of pulmonary amoebiasis under the heading "Haematogenous pulmonary

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abscess without liver involvement". They noted that the low mortality rate reported in these patients was undoubtedly due to the fact that the majority received emetine. In their personal series of cases there were no haematogenous pulmonary abscesses.

In this presentation of pulmonary amoebiasis the symptomatology and clinical findings are confined to the chest. Local signs may be found over any aspect of the chest and need not necessarily be found at the lung base as in hepatothoracic amoebiasis. The contents of these haematogenous abscesses have been said to be of a purulent nature and not of an anchovy sauce or chocolate sauce character (Daniels and Childress, 1956). Others who have reported this presentation of pulmonary amoebiasis have been Ramond et al. (1923), Hameed (1945), Chaudhuri and Chaudhuri (1946), Sullivan and Bailey (1951), Deaton and Garrett (1956).

There were no examples of haematogenous pulmonary lesions without hepatic involvement in this study. It may be impossible on clinical grounds to rule out a hepatic origin for these lung lesions as necropsy may reveal a "silent" liver abscess not detected clinically (Mansen-Bahr, 1944; Verner, 1948), or show evidence of a healed amoebic liver abscess (Opie, 1901). Finally, following extension into the lung a liver abscess may decrease in size leaving little or no evidence of hepatic involvement (Abdel-Hakim and Higasi, 1958).

Presuppurative Lesions: In the absence of clinical evidence of hepatic amoebiasis pneumonic shadows or infiltration of amoebic origin may occur in any part of the lung fields (Blanc and Siguier, 1946). In such presentations haematogenous spread from the bowel has been postulated and response to specific therapy in the form of emetine noted (Blanc and Siguier, 1946; Dell Aquila, 1949; Heinemann, 1956).

Transient, migrating radiological pulmonary infiltration with eosinophilia, characteristic of Loeffler's syndrome have been documented as presentations of pulmonary amoebiasis (Neff and Mason Hicks, 1942; Randall, 1945). The etiological relationship in these patients was considered on the basis of finding E.histolytica in the stools, and on the response to specific therapy.

There were no examples of isolated pulmonary infiltrations or presentations which could be described as Loeffler's syndrome in this study. From the literature it seems that amoebic pulmonary infiltration, although a rarity, is possible on the basis of haematogenous spread from the large bowel. The transient pulmonary infiltrations of Loeffler's syndrome have been observed in association with a wide range of intestinal parasites, drug sensitisation, and in the condition tropical eosinophilia (Harrison, 1958). In my opinion further evidence is required before including the amoeba in this list.

Amoebic Bronchitis: In 1923 Petsetakis reported 2 patients whom he

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considered to be suffering from amoebic bronchitis on the grounds that amoeba were identified in the sputum and there was a satisfactory response to specific treatment. Surprisingly he claimed to have identified cysts of E.histolytica in the sputum of one of these patients. A year later Panayotatou and Netter (1924) reported a patient suffering from true primary amoebic bronchitis, in that inhalation of the amoeba was the postulated pathogenic basis. Other examples of amoebic bronchitis have been reported by Yalouzis (1925), Haberfeld (1927), Motta (1938), Rizzo (1939), Huber (1950), Wasielewski et al. (1951) and Abdel-Hakim and Higazi (1956).

Although there were patients shown to have right basal bronchiectasis following suppurative amoebic lesions in this study, the isolated entity of amoebic bronchitis was not encountered. As yet a pathological basis has not been shown to exist for this doubtful clinical entity, and workers have not been able to reproduce the condition in experimental animals (Carvaillo and Sautet, 1926). A study of case reports in the literature suggested that in some instances patients labelled as amoebic bronchitis could have been suffering from hepatothoracic amoebiasis.

It has been suggested that the amoeba seen by Petzetakis and others may have been Entamoeba gingivalis (Deschiens and Helmotte, 1928). Iron-haematoxylin staining procedures are necessary to distinguish this non-pathogenic non-haematophagous commensal of the

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buccal cavity from E.histolytica (Sutliff et al., 1951). At this hospital patients are seen who harbour E.gingivalis in their mouths and E.histolytica in their stools. The above are but few of the queries which may be levelled against the existence of the clinical entity amoebic bronchitis.

Bronchial Asthma and Amoebiasis: Amongst 120 patients in the Argentine with E.histolytica in their stools Carri (1948) found 12 to be suffering from respiratory allergies. Three of these experienced asthmatic episodes which resisted conventional treatment and it was claimed that improvement was maintained following treatment with emetine and yatren. More recently Hamilton and Lutwyche (1960) reported a patient suffering from asthmatic-like episodes with cysts of E.histolytica in the stools who also responded to specific therapy. Others who have claimed an association between the amoeba and bronchial asthma have been Stefano (1939), Rodrigues (1945) and Huber (1950).

Too often in the above rarely reported and doubtful presentations of pulmonary amoebiasis diagnostic claims have been made on the basis of response to specific treatment. The dangers and difficulty of forming diagnostic conclusions on such a basis, are well recognised (Deschiens and Melnotte, 1928; Wilmot, 1962). The asthmatic who is frequently highly susceptible to suggestion is a most unsuitable patient in this respect. At this hospital, where hundreds of patients with intestinal amoebiasis and bronchial asthma are seen annually, no

/association ...

association between the two conditions has been noted.

There were no patients in this study in whom the amoebic process involved the thoracic cage. Reports of this complication have been documented by Wilmot (1949), Bell et al. (1954), Diamond and Scribner (1956), and indicate that involvement of the thoracic cage results from direct extension of an amoebic liver abscess or amoebic empyema.

CONCLUSIONS

The clinical presentation in hepatothoracic amoebiasis is fairly characteristic and is similar in many respects to that of an uncomplicated amoebic liver abscess. Pain situated in the right lower chest and right upper quadrant of the abdomen, frequently referred to the right shoulder, is the most constant symptom. Associated symptoms may be fever, sweating, a non-productive cough or cough productive of large quantities of reddish-brown or blood-stained pus, and dyspnoea.

On examination a low-grade pyrexia, evidence of recent weight loss, pale mucosae and clubbing of the fingers may be seen. The sputum varies in character from typical anchovy sauce or chocolate coloured sputum to blood-stained or yellow pus. There may be frank haemoptysis. The sudden production of large quantities of anchovy sauce sputum suggests a hepatobronchial fistula; when bile-stained it is pathognomonic of a bronchobiliary fistula.

On local examination signs are almost invariably found in

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the right lower chest and right upper quadrant of the abdomen. The thoracic findings may suggest pulmonary or pleural involvement or an elevated right diaphragm. It is often impossible from the local findings to ascertain the nature of the parenchymal lesion or to make the distinction between a small or moderate sized effusion and an elevated right diaphragm. Tenderness and guarding and a tender, enlarged liver are frequently found in the right upper quadrant. Tender hepatomegaly, although a most significant finding, is not invariably present. Less commonly the left lower chest may be the site of the lesion and the abdominal findings those of a left lobe liver abscess.

Presentations have been reported in which signs of a liver abscess are associated with pulmonary signs in situations remote from the diaphragm. Such presentations, in which haematogenous spread from the liver abscess has been postulated, are rare and there were no examples in this study.

The terms presuppurative and non-suppurative pleural or pulmonary amoebiasis have been used to describe presentations in which clinical and aspirational evidence of suppuration is absent, but which in other respects resemble the classic presentation mentioned above. A number of examples of presuppurative amoebic pleural effusions were seen in this study.

Very rarely haematogenous pulmonary amoebiasis may present

/with ...

with signs in any aspect of the chest in the absence of signs of an amoebic liver abscess. There were no such presentations in this study.

Although E.histolytica has been incriminated in the etiology of Loeffler's syndrome, bronchitis and bronchial asthma, there were no such presentations seen in this study and more conclusive evidence is required before an etiological relationship can be accepted in these conditions.

INVESTIGATIONS IN PLEUROPULMONARY AMOEBIASIS

RADIOLOGICAL FINDINGS

Amoebic Liver Abscess: Radiological changes are present in about 75 per cent of all cases of amoebic liver abscess. These consist of elevation, deformity, immobility or reduction in movement of the diaphragm and linear atelectasis or patchy opacities at the lung base, usually on the right side and infrequently on the left (Wilmot, 1962).

The interpretation of basal pulmonary shadows associated with an amoebic liver abscess may be difficult. Linear plate-like shadows suggesting atelectasis and confluent or patchy shadows suggesting pneumonia, bronchopneumonia or pneumonitis, have been reported (Munk, 1944; Isaac, 1945; Blanc and Siguier, 1946; Coirault et al., 1955; Lamont and Peeler, 1958). Munk considered that the linear or plate-like shadows represented areas of atelectasis resulting from the mechanical effects of the elevated or immobile right diaphragm. He was of the opinion that many of these basal shadows diagnosed as pneumonic consolidation are, in fact, due to atelectasis (Munk, 1944). Others have suggested that these changes may at times represent the presuppurative stage of pulmonary amoebiasis (Blanc and Siguier, 1946; Coirault et al., 1955), or, in some instances, are the result of an associated bacterial pneumonia or pneumonitis (Blanc and Siguier, 1946).

The radiological changes mentioned above are those which may be seen with an uncomplicated liver abscess. Their similarity to the

/radiological ...

radiological change reported in this study is obvious. They have been mentioned to illustrate that it may be impossible on radiological grounds to determine whether direct extension of a liver abscess into the chest has occurred or whether the changes are simply those produced by an uncomplicated liver abscess.

Pleuropulmonary Amoebiasis: Ochsner and DeBakey (1936) stated that the radiological examination may be quite characteristic following the perforation of an amoebic liver abscess into the lung. Prior to rupture the liver abscess may produce a localised bulging of the diaphragm which following rupture is replaced by a triangular shadow with its base towards the liver. This shadow is best seen on the lateral films. Others who have described similar changes following extension of a liver abscess into the right lung base have been Hughes and Westphal (1947), Chakravarti (1951,1952,1953) and Kilgore (1951). None of these workers, however, has offered a satisfactory explanation as to what these triangular shadows represent. The circumscribed semicircular shadows seen at the base of the right lung in 8 of my patients were probably similar in origin to the triangular shadows reported by Ochsner and DeBakey and others mentioned above. In my opinion these shadows are probably produced by the inflammatory reaction surrounding a hepatobronchial fistula or a hepatopulmonary abscess. In 3 of the 8 patients with circumscribed semicircular consolidation the area of consolidation was subsequently replaced by cavitation. This was

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regarded as further evidence that these shadows represent an inflammatory reaction in the vicinity of a hepatobronchial fistula or hepatopulmonary abscess.

Following extension of a liver abscess, Alarcon (1943,1954) described a crescent-shaped shadow superimposed on the liver silhouette which was smaller and less dense than the liver shadow. This he considered pathognomonic of extension of a liver abscess into the right lung. It is likely that these crescent-shaped shadows are similar in origin to the triangular shadows described by Ochsner and DeBakey (1936) and the circumscribed semicircular shadows seen in this study.

A number of authors have described a "string-like shadow" proceeding vertically from a localised bulge of the diaphragm to a pneumonic shadow in the lung (Flynn and Warren, 1928; Schorr and Schwartz, 1951; Abdel-Hakim and Higazi, 1958). Abdel-Hakim and Higazi (1958) considered that, as these linear shadows were observed in cases of proved amoebiasis and disappeared after anti-amoebic treatment, it may be assumed that they result from spread of the inflammatory process. These string-like shadows were not seen in the radiographs of my patients and in my experience are an uncommon radiological finding in pulmonary amoebiasis.

The experience of a number of workers has been that consolidation at the right base, either patchy or bronchopneumonic, confluent or lobar, is one of the most frequent radiological changes resulting from

/extension ...

extension of a liver abscess into the right lung (Beckless, 1950; Chakravarti, 1951,1952,1953; Lamont and Pooler, 1958; Takaro and Bond, 1958). In the less common presentation of pulmonary amoebiasis following haematogenous spread single or multiple foci of consolidation may occur in any aspect of the lung fields (Blanc and Siguier, 1946; Ceirault et al., 1955). In the patient reported by Deaton and Garrett (1956) consolidation progressing to cavitation was seen in the right upper lobe. Cromnier and Darbon (1952) reported a patient with radiological changes similar to those seen with multiple secondary deposits in the lung fields.

Two-thirds of the patients with pulmonary amoebiasis in this study presented with consolidation in the right lower lung field. Three varieties were seen. The first, consisted of an area of circumscribed semicircular consolidation with the base of the semicircle perched on the right diaphragm; the second, of lobar consolidation of the lower or occasionally the middle lobe; and the third, of patchy consolidation in the right lower lung field. In my experience consolidation in a situation other than the right lower lung field is rare in pulmonary amoebiasis. There were only 2 examples in this study, one (case 6) in the left lower lobe, the other (case 3), in the left upper lobe. It is likely that in many instances the radiological consolidation seen in pulmonary amoebiasis represents areas of intrapulmonary suppuration. This was shown at necropsy in 2 patients (cases 3 and 6) in this study and was suggested in a further 3 when areas of radiological

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consolidation were subsequently replaced by cavities with air-fluid levels.

Amoebic consolidation may progress to abscess formation (Ceirault et al., 1955; Druckman and Schorr, 1944). The most frequent situation of these abscesses is in the right lower lung field (Ochsner and DeBakey, 1936; Takaro and Bond, 1958; Wilmot, 1962). Dorner (1945) stated that these basal abscesses are nearly always anterior in position and that prior to their formation there may be an upward bulging of interlobar septum. Occasionally, single or multiple abscesses may be seen in situations remote from the diaphragm, a finding which suggests a haematogenous origin (Ochsner and DeBakey, 1936; Chapman et al., 1948; Chatterjee and Sen Gupta, 1949; Lindsay et al., 1951; Deaton and Garrett, 1956).

Twelve of the 30 patients with pulmonary amoebiasis in this study had radiological evidence of cavitation of the lower lobe of the right lung. Six of these 12 presented with consolidation in this situation which subsequently progressed to cavitation. In my experience cavitation is a common radiological finding in pulmonary amoebiasis and the cavity is nearly always situated in the basal aspect of the lower lobe of the right lung. Radiological cavitation in a situation other than the basal aspect of the right lower lobe was seen in only 2 of my patients, in both, the apical segment of the right lower lobe was involved. Cavitation in situations other than the right base was seen at necropsy in 2 patients (cases 3 and 6) in this study. In one (case 3) multiple

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cavities were seen in the upper lobe of the left lung, in the other (case 6) an abscess cavity was found in the basal aspect of the left lower lobe.

Druckman and Scherr (1944) described a sequence of five radiological changes which may be seen with extension of a liver abscess into the lung. The first, consisted of elevation of the right diaphragm; the second, of blurring of its outline and clouding in the right costophrenic angle; the third, of patchy or band-like shadows in the base of the right lung; the fourth, of confluent shadowing in the right lower lobe simulating pneumonia; and finally, cavitation of this confluent shadow. Frequent serial radiographs would be necessary during the stage of extension of the liver abscess into the chest before the above radiological sequence could be observed in its entirety. There were therefore few patients in this study in which this sequence was seen. Patients were seen, however, with radiological changes similar to each of the stages proposed by Druckman and Scherr (1944) and in my opinion their proposed radiological sequence can be reasonably accepted as an order of events likely to be seen in pulmonary amoebiasis.

The majority of amoebic empyemas occupy the right pleural space, they are usually large, produce mediastinal displacement, and are sometimes encysted (Verges and Hermenjat-Gerin, 1932; Huard and Meyer-May, 1936; Lament and Peeler, 1958). An amoebic empyema situated in the left chest is a rare occurrence; examples have been reported by

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Gosh (1954), Lamont and Pooler (1958), Abdel-Hakim and Higasi (1958). Fifteen of the 17 amoebic empyemas seen in this study were situated in the right chest and only 2 (cases 6 and 40) were seen in the left chest. These empyemas were nearly always large effusions and mediastinal displacement was common. In 3 patients (cases 44,45 and 47) the effusions appeared to be encysted and in 4 air-fluid levels in the pleural space associated with a cough productive of pus suggested the possibility of a bronchopleural fistula. Recently Whittaker (1963) stated that direct extension of an amoebic empyema into the pleural space is uncommon and that before this occurs the abscess usually tracks up the greater fissure. In the 3 patients (cases 41,43 and 48) in this study who developed right-sided empyema there was no evidence of this radiological sequence.

Serous or serosanguineous effusions of amoebic origin have been reported by Anagnostopoulos (1940), Blanc and Siguier (1946), Coirault et al. (1955) and others. These are usually situated in the right chest, are small to moderate in size and may be encysted or have an interlobar situation. Carruthers (1947) reported patients with bilateral serous effusions which he considered to be of amoebic origin. Radiologically the serous and serosanguineous effusions in this study were small effusions occupying the right costophrenic angle. A number of patients were seen with radiological changes of a small effusion occupying the base of the right greater fissure. These effusions were

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considered to be presuppurative rather than suppurative in nature because of their small size and rapid response to treatment. There were no patients who presented with bilateral effusions in this study. Such a presentation is difficult to explain unless one postulates the existence of concomitant right and left-sided liver abscesses both involving their respective pleural cavities. One patient (case 48) was seen who initially developed a right-sided effusion associated with a subphrenic abscess and subsequently developed a left subphrenic abscess and pleural effusion.

Wilmot (1962) has emphasised that in pulmonary amoebiasis particular attention should be paid to the position and contour of the right diaphragm, as radiological evidence of abnormality of this structure provides valuable information concerning the hepatic origin of the lung lesion. Screening of the chest may be helpful in determining its position and mobility. Local or generalised elevation of the right diaphragm was seen in 37 of the 50 patients with pleuropulmonary amoebiasis in this study. In only one instance (case 40) did radiographs show elevation of the left diaphragm. An elevated or deformed right diaphragm was a most important radiological finding in my patients as it provided evidence of a subphrenic source for the thoracic lesion. There were a few patients in whom air-fluid levels were seen below the right diaphragm prior to needle aspiration of the liver abscess. This finding was considered pathognomonic of a hepatobronchial fistula.

Less frequently employed diagnostic radiological procedures in pleuropulmonary amoebiasis have been, barium meal to localise a left lobe amoebic liver abscess (Allman et al., 1961), the injection of contrast media into the bronchi to demonstrate a hepatobronchial fistula (Cannavo and Cola, 1931; Ochsner and DeBakey, 1936; Hughes and Westphal, 1947) and the injection of air into the peritoneal cavity to demonstrate hepatodiaphragmatic irregularities (Cannavo and Cola, 1931; Clark and Dutta, 1945). A left lobe liver abscess was demonstrated by a barium meal in one patient (case 6) in this study and barium studies may be helpful in localising a deep-seated left lobe liver abscess. Attempts to outline hepatobronchial fistulae with contrast media were unsuccessful in my patients, but residual right basal bronchiectasis and evidence of pleural thickening were demonstrated by bronchography.

It can be seen from the above review and discussion that the reported radiological changes in pulmonary amoebiasis are many and varied. To conclude this review there follows a brief summary of the changes which are worthy of note.

Common pulmonary changes are areas of consolidation situated in the right lower and middle lobes. The left lung is seldom involved. The consolidation may be lobar or patchy or take the form of areas of circumscribed semicircular consolidation or crescent-shaped shadows perched on the right diaphragm. Serial radiographs may show cavities replacing these areas of consolidation. Cavitation is a

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fairly frequent finding and the cavity is most commonly situated in the basal aspect of the right lower lobe. Associated with either consolidation or cavitation there may be changes suggestive of atelectasis at the right base. Occasionally in patients with hepatobronchial fistulae there are no radiological changes.

Amoebic pleural effusions are nearly always found in the right chest and may range in size from small effusions in the costophrenic angle to massive effusions producing mediastinal shift. Occasionally these effusions are encysted or have an interlobar position spreading the base of the greater fissure. An air-fluid level in a pleural effusion associated with a productive cough is suggestive of a bronchopleural fistula.

Any combination of the above pleural and pulmonary changes may be seen and a most important associated finding is an elevated or deformed right diaphragm. This should always suggest a possible subphrenic origin of the right basal lesion. An air-fluid level beneath the right diaphragm in a patient with a cough productive of pus is pathognomonic of a hepatobronchial fistula. Screening of the chest may show immobility of the diaphragm.

HAEMATOLOGICAL FINDINGS

Haemoglobin: Anaemia is a recognised finding in amoebic liver abscess. It is usually of a normocytic normochromic variety and may be severe when abscesses are large or the history is long (Lamont and Pooler, 1958; Wilmot, 1962). Alarcon (1954) regarded the anaemia in pulmonary

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amoebiasis to be related to the degree of associated liver destruction. I think it more likely that the severity of the anaemia is related to the duration of the illness and the extent of the hepatothoracic suppuration. Moderate to severe anaemia of a normochromic normocytic type was frequently present in my patients. The impression formed was that extension of the amoebic process beyond the confines of the liver increased the likelihood of a severe associated anaemia. In the few patients in whom serum iron levels, total plasma iron-binding capacity and bone marrow iron stores were estimated the findings were similar to those reported in the anaemia of infection by Stevens et al. (1953). There was no evidence that haemolysis was operative in the pathogenesis of the anaemia, a possibility suggested by Lamont and Pooler (1958).

White Cell Count: A moderate leucocytosis is frequently present in patients with pleuropulmonary amoebiasis (Alarcon, 1954; Webster, 1960). Ochsner and DeBakey (1936) noted that the leucocyte count and polymorph percentage are not so high in patients with amoebic infection of the lung as with corresponding pyogenic infections. They stated that in cases of pulmonary amoebiasis accompanied by high leucocyte and polymorphonuclear counts secondary pyogenic invasion should be suspected. In my experience the white cell count in pleuropulmonary amoebiasis may be within normal limits but is usually moderately increased and rarely greatly increased. Because of this variability the white cell count is of limited value.

The Sedimentation Rate: The erythrocyte sedimentation rate in hepatic amoebiasis is of use in determining the progress of the disease and response to treatment rather than of diagnostic significance (Klatskin, 1946). In my patients an increase in the erythrocyte sedimentation rate was a constant finding and recovery was accompanied by a decrease in the rate.

EXAMINATION OF THE SPUTUM

The naked eye characteristics of amoebic sputum have been discussed in the section dealing with the clinical findings. Councilman and Lafleur (1891) noted that on microscopic examination, red blood cells, leucocytes, round alveolar epithelial cells, degenerate liver cells and elastic fibres are the cellular elements which may be seen in the sputum. They stated that amoeba are constantly present. Other workers have reported the difficulty which may be experienced in finding trophozoites of E.histolytica in the sputum of patients with suspected pulmonary amoebiasis (Manson-Bahr, 1923; Ochsner and DeBakey, 1936; Dorner and Friedlander, 1941; Beckless, 1950; Takaro and Bond, 1958). To date, Chakravarti (1951,1952,1953) has been the most successful in this respect, having identified the parasite in the sputum of 11 out of 26 patients with pulmonary amoebiasis. He emphasised the necessity of examining freshly prepared warm saline preparations and herein lies the probable explanation of his success.

Kofoid and Swezy (1924) compared the morphology of E.histolytica

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and E.gingivalis, a non-pathogenic, non-haematophagous, amoeba of the buccal cavity. Deschiens and Melnotte (1928) suggested that this amoeba which could be found in the sputum might be thought to come from the bronchi or lungs. They advised an examination of material from the mouth to exclude this error. More recently, Sutliff et al. (1951) reported 2 patients in whose bronchial aspirate and sputum, E.gingivalis was identified. They stressed the importance of examining smears stained with iron-haematoxylin to differentiate E.histolytica from E.gingivalis, particularly in patients with no evidence of associated hepatic amoebiasis.

Bernard (1929) stated that in the early stages of pulmonary amoebiasis there may be few bacteria in the sputum, but that later, as a result of secondary infection, many organisms may be present. Microscopy may also reveal striated muscle fibres from the diaphragm or liver cells. (Manson-Bahr, 1943; Islam et al., 1960). Examination of the sputum for bile may be rewarding in patients suspected of having a bronchobiliary fistula (Webster, 1960). E.histolytica was identified in the sputa of 10 of the 30 patients with pulmonary amoebiasis in this study.

When fresh specimens of sputa or aspirates from my patients were immediately examined by experienced workers in the local Amoebiasis Research Unit, E.histolytica was frequently identified. When, however, specimens were sent to the general laboratory of this hospital, where there was usually a considerable delay prior to examination, the

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parasite was never found. It is of the utmost impertance that a fresh specimen of sputum be examined. The majority of the E.histolytica isolated from the sputum of my patients were haematophagous. When difficulty was experienced in distinguishing those that contained no red cells from E.gingivalis, iron haematoxylin stains were used.

In only a few instances did culture of the sputa result in heavy growths of bacterial pathogens and in no patients were acid-fast bacilli seen or cultured.

PLEURAL ASPIRATION

"The aspiration of pleural fluid is an invaluable diagnostic procedure in suspected pleural amoebiasis and the finding of characteristic chocolate sauce pus, especially if it contains amebae, is pathognomonic" (Ochsner and DeBakey, 1936). Although the above statement is true of a percentage of patients with pleural amoebiasis, Takaro and Bond (1958), in their review of the literature, found in 105 amoebic aspirates from the pleura, liver and pericardium, that less than half were of the characteristic chocolate sauce variety. It is important not to dismiss thoracic amoebiasis as a diagnostic possibility simply because characteristic pus is not obtained.

Although amoebic pus aspirated from the pleural cavity is usually bacteriologically sterile (Geirault et al., 1955), it should always be cultured to exclude secondary bacterial invasion (Wilmot, 1962). It has been stated that amoeba are infrequently found in the aspirated pus (Ochsner and DeBakey, 1936).

Occasionally serous or seresanguineous fluid is aspirated from the pleural cavity in patients with hepatic amoebiasis (Blanc and Seguier, 1946; Carruthers, 1947; Ceirault et al., 1955). The pathogenesis of this variety of pleural amoebiasis has been discussed. In the patient reported by Damade and Lavignolle (1947) the fluid was an exudate and contained equal numbers of polymorphonuclear cells and lymphocytes as well as endothelial cells. Reviewing the literature, I was unable to find any instances in which E.histolytica had been identified in serous effusions associated with hepatic amoebiasis.

Characteristic anchovy sauce pus was aspirated from the pleural space in a number of my patients, but serous or seresanguineous fluid and yellow or blood-stained pus, were also obtained. This finding is in accord with the experience of Takaro and Bond (1958), and others mentioned above.

Trophozoites of E.histolytica were identified in the pus of 6 of the 15 patients with amoebic empyemas in this study. In the large majority the pus was sterile on culture for bacteria and acid-fast bacilli were never found.

The serous and seresanguineous effusions in my patients had protein levels compatible with an exudate and contained variable numbers of red blood cells, polymorphonuclear cells and lymphocytes. They were invariably sterile on culture for bacteria and acid-fast bacilli.

LIVER ASPIRATION

The diagnostic value of needle aspiration of the liver in patients with suspected amoebic liver abscess has been repeatedly emphasised (Ochsner and DeBakey, 1943; Lamont and Peeler, 1958; Wilmot, 1962). Although typical anchovy or chocolate sauce pus will frequently be obtained, this is by no means invariable, as the aspirate of an amoebic liver abscess may show a wide range of colour and consistency (Berne, 1942; Takaro and Bond, 1958; Lamont and Peeler, 1958).

Ochsner and DeBakey (1936) stated that in suspected pleuropulmonary complications of amoebic liver abscess, aspiration of the abscess is justified. More recently, Abdel-Hakim and Higazi (1958) reporting 28 patients with pleuropulmonary amoebiasis, made no mention of this procedure as an aid to diagnosis and in other recent publications its value has been insufficiently stressed (Takaro and Bond, 1958; Singh and Jolly, 1962). Keeley et al. (1962) proposed aspiration biopsy of the liver as a useful diagnostic procedure in patients with suspected amoebic liver abscess. Needle aspiration has proved a satisfactory diagnostic procedure in hepatic amoebiasis and I believe aspiration biopsy is not warranted.

Aspiration of pus from below the diaphragm established the source of the pleural or pulmonary lesions in 14 of the 50 patients in this study. The procedure was therefore of diagnostic value. From 8 patients typical anchovy sauce pus was obtained, whilst in the remaining 6 the character of the pus was variable. Aspiration biopsy was not

/employed ...

employed as a diagnostic procedure in my patients.

The dangers of haemorrhage or bacterial contamination in liver aspiration have been noted (Huard and Meyer-May, 1936; Wilmot, 1962). In my experience when the technique and precautions recommended by Wilmot (1962) are employed, serious complications are rare.

STOOL EXAMINATION

Conflicting views exist as to the diagnostic significance of E.histolytica identified in the stools of patients with suspected pulmonary amoebiasis. Chakravarti (1951), working in an endemic area, maintained that E.histolytica were so frequently found in the stools of his patient population that their presence was of little diagnostic significance. In more temperate climates, where hepatic and pleuro-pulmonary amoebiasis occur sporadically the importance of examining the stools for E.histolytica in suspected pleuropulmonary amoebiasis has been stressed (Kilgore, 1951; Daniels and Childress, 1956; Webster, 1960). In some presentations of pulmonary amoebiasis, particularly the so-called primary variety, evidence of intestinal amoebiasis may be the only pointer to the etiology of the pulmonary lesion (Manson-Bahr, 1944).

In this study 6 (12 per cent) patients had associated clinical features of amoebic dysentery, 13 (26 per cent) admitted to a past history of dysentery, and E.histolytica were found in the stools of 7 (14 per cent) patients. In my opinion, evidence of intestinal amoebiasis should arouse suspicion of a possible amoebic etiology in patients with pulmonary

/pathology ...

pathology of undetermined origin. The absence of evidence of intestinal amoebiasis in such patients, however, by no means excludes an amoebic etiology.

COMPLEMENT FIXATION TEST

Isar (1914) and Craig (1927) were among the first to suggest that a complement fixation test might be useful in the diagnosis of intestinal and hepatic amoebiasis. In recent years it has been suggested that this test may be of value in the diagnosis of pleuropulmonary amoebiasis (Kilgore, 1951; Coman et al., 1950). Not all workers using this test, however, have obtained the consistent results reported by Craig in 1939 (Paulson and Andrews, 1938; Magath and Meleney, 1940), one of the main difficulties being the preparation of a suitable antigen (Hussey and Brown, 1950). Kladon-Dew and Maddison (1952) found that only a proportion of those who harbour E.histolytica in their bowel show complement fixing antibodies but when liver invasion occurs these antibodies are nearly always present.

Recently Maddison (1963) studied the pattern of antibody response to tissue invasion with E.histolytica by means of a gel-diffusion precipitin test and found this technique to be more sensitive than the complement fixation test used by Craig (1939), Magath and Meleney (1940), Dolkart et al. (1951) and Kladon-Dew and Maddison (1952), but less sensitive than the haemagglutination test of Kessel et al. (1961).

The complement fixation test was not employed in this study. The gel-diffusion and haemagglutination tests mentioned above were done in 10 of my patients and in each instance positive results were obtained. Further work will have to be done to assess the value of these tests in the diagnosis of pleuropulmonary amoebiasis as it seems that they are likely to prove of diagnostic value.

LIVER FUNCTION TESTS

Powell (1959) reviewed the literature concerning the value of liver function tests in the diagnosis of hepatic amoebiasis. He showed, in a series of his own patients, that although changes occur, these are not distinctive enough to be of diagnostic significance. Liver function tests were of no diagnostic assistance in this study and in my experience are seldom of value in the diagnosis of hepatic or pleuropulmonary amoebiasis at this hospital.

CONCLUSIONS

Chest radiographs are among the most important investigations in pleuropulmonary amoebiasis. Radiological changes are nearly always seen at the right lung base. Changes such as linear shadows suggesting atelectasis, semicircular areas of consolidation perched on the right diaphragm, patchy or lobar consolidation, abscess formation and pleural effusions may be present. Localised or generalised elevation of the right diaphragm is an important finding and screening may show immobility of the diaphragm. Radiological changes are uncommon

/at ...

at the left base and rarely present in the remaining lung fields.

Moderate to severe anaemia of a normocytic normochromic type may be found in patients with pleuropulmonary amoebiasis. Moderate elevation of the white cell count is usual but not invariable. The sedimentation rate is almost invariably elevated.

Difficulty may be experienced in finding E.histolytica in the sputum of patients with pleuropulmonary amoebiasis and fresh specimens should always be examined. E.gingivalis, a non-haematophagous commensal of the buccal cavity, may be mistaken for E.histolytica. This error can be avoided by examining iron-haematoxylin stained smears. Significant growths of pathogenic bacteria are seldom obtained from culture of amoebic sputum.

Pleural aspiration is an invaluable investigation in suspected pleural amoebiasis. Serous, serosanguineous, typical anchovy sauce pus, blood-stained pus or yellow pus may be aspirated. A fresh specimen of the aspirated pus should be examined forthwith for trophozoites of E.histolytica. The pus should be examined and cultured for pyogenic and acid-fast bacteria. Pyogenic organisms are seldom cultured from amoebic pleural aspirates.

Aspiration of a suspected liver abscess is an important investigation in patients with suspected pleuropulmonary amoebiasis as when successful it establishes the source of the thoracic pathology. Pus from an amoebic liver abscess may vary in character and need not always be of a typical anchovy or chocolate sauce colour. Fresh

/specimens ...

specimens should be examined forthwith for amoeba and the pus cultured for pyogenic bacteria.

The stools of patients with suspected pleuropulmonary amoebiasis should be examined for E.histolytica.

Doubt still exists as to the value of the complement fixation test, haemagglutination test and gel-diffusion test as investigations in hepatic and pleuropulmonary amoebiasis. Findings in this study suggested that the haemagglutination test and the gel-diffusion test may prove of value in the diagnosis of thoracic amoebiasis but more experience with these tests is required to confirm this impression.

THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF PLEUROPULMONARY AMOEBIASIS

HEPATOTHORACIC AMOEBIASIS

It has been emphasised that a knowledge or awareness of the existence of pleuropulmonary amoebiasis is an important factor in diagnosis (Ochsner and DeBakey, 1936; Hughes and Westphal, 1947; Sullivan and Bailey, 1951).

Councilman and LaFleur (1891) referring to the diagnostic significance of the sputum coughed up in pulmonary amoebiasis stated, "There is no matter like it expectorated in any disease of the lung itself, and I believe its appearance is pathognomonic of an abscess of the liver or at least of an abscess perforating the lung. I have been led by it to detect an abscess of the liver of which I had previously no suspicion".

Ochsner and DeBakey (1936) noted that when a history of previous diarrhoea, moderate elevation of the temperature and enlargement and tenderness of the liver are associated with pulmonary manifestations, amoebic infection of the lung should be suspected. They also emphasised the diagnostic import of the coughing up of large quantities of chocolate sauce pus. Others who have noted the diagnostic value of a tender hepatomegaly associated with right basal chest signs in hepatothoracic amoebiasis have been Kilgore (1951), Chakravarti (1951,1952,1953), Abdel-Hakim and Higazi (1958) and Tandon and Khanna (1960).

Opposing the above views Langston and Fox (1947) stated that no typical picture or syndrome can be described in pleuro-pulmonary amoebiasis whilst Bookless (1950) noted that in his patients signs and symptoms of chest disease were often acute and preceded those of the underlying liver condition (Bookless, 1950).

The consistency of the clinical presentation in the majority of my patients was helpful in establishing the diagnosis. Symptoms and signs referable to the right lower chest associated with a tender hepatomegaly or guarding and tenderness in the right upper quadrant were suggestive, particularly when accompanied by a cough productive of large quantities of blood-stained or anchovy sauce pus. A cough productive of typical anchovy sauce pus was virtually diagnostic. Not all my patients, however, produced this characteristic type of sputum and in some instances it was the large quantity, rather than the quality of the sputum, which suggested the diagnosis. Only 60 per cent of patients in this study presented with a tender hepatomegaly, but in the absence of this sign guarding and tenderness in the right upper quadrant were usual. The association of guarding and tenderness in the right upper quadrant of the abdomen and right shoulder tip pain was a combination which suggested the diagnosis of an amoebic liver abscess in my patients. In a small percentage of patients concomitant dysenteric symptoms or a past history of dysentery were regarded as circumstantial evidence for the diagnosis. It is apparent that the clinical features of diagnostic value in my patients were similar to those reported by Ochsner and DeBakey (1936), Chakravarti (1951),

/Kilgore ...

Kilgore (1951) and others mentioned above and that in the majority a fairly characteristic clinical presentation was encountered.

Atypical presentations seen in this study although few in number, are worthy of mention. There were a few patients in whom there were no symptoms and signs referable to the right upper quadrant of the abdomen. Fortunately in most of these features such as the character and quantity of the sputum or an elevated or immobile right diaphragm suggested the source of the thoracic lesion. Clinicians with little or no experience of pleuropulmonary amoebiasis could easily overlook the subphrenic lesion in such presentations, and fail to make the diagnosis. In a sense left basal hepatopulmonary amoebiasis may be regarded as an atypical presentation because of its infrequent occurrence and the tendency by many to regard hepatopulmonary amoebiasis as a right basal syndrome. I have seen patients in whom there was a delay in diagnosis and those in whom the diagnosis was missed because of the above factors. There was one such patient (case 6) in this study who presented with haemoptysis, signs at the base of the left lung and severe anaemia.

Singh and Jelly (1962) stated that in their experience radiological findings constituted the most important investigation in establishing the diagnosis of hepatopulmonary amoebiasis. Others have stressed the importance of obtaining lateral radiographs of good quality as well as postero-anterior views (Geirault et al., 1955).

Radiology was a most useful aid in the diagnosis of pulmonary amoebiasis in this study. Diagnostically the most important feature was an elevated or deformed right diaphragm, a finding which focused attention on a possible subphrenic source of the changes seen at the base of the right lung. In many patients the pulmonary changes at the right base were non-specific, the most distinctive of these being the circumscribed semicircular areas of consolidation adjacent to the right diaphragm.

With the exception of an elevated right diaphragm there were no distinctive radiological features in my patients with amoebic pleural effusions. Unfortunately pleural effusions sometimes obscured the position of the right diaphragm which was only seen to be elevated after aspiration or absorption of the fluid.

In patients with suspected hepatothoracic amoebiasis immobility of the right diaphragm seen on screening the chest may be regarded as evidence for the existence of a subphrenic abscess. This procedure should probably have been used more frequently in this study.

Although techniques such as bronchography, pneumoperitoneum and the injection of contrast media into abscesses or the pleural cavity may be useful in isolated instances, they are seldom essential for the diagnosis. I believe it wise, because of the danger of secondary bacterial contamination, to avoid, as far as possible, the injection of foreign substances into abscesses or pleural cavities of patients

/with ...

with hepatothoracic amoebiasis.

The diagnostic value of pleural aspiration has been stressed and in this study was most useful in establishing the nature of the pleural fluid. Typical sterile anchovy sauce pus when aspirated was very suggestive, whilst the identification of E.histolytica in the aspirate confirmed the diagnosis. In those patients in whom the aspirate was not typical and the parasite was not identified the diagnosis was based on clinical, radiological or aspirational evidence of an associated amoebic liver abscess. Coirault et al. (1955) stressed the importance of not overlooking the underlying liver abscess when serous fluid is aspirated from the pleural cavity of patients with non-suppurative amoebic effusions.

The aspiration of pus from an amoebic liver abscess was of diagnostic value in a number of my patients in that it confirmed the suspected source of the right basal thoracic lesion. In my opinion the majority of workers have made insufficient use of this most helpful diagnostic procedure.

The diagnostic significance of a past history of dysentery and examination of the stools for E.histolytica in pulmonary amoebiasis has been previously discussed.

Absolute confirmation of the clinical diagnosis of hepatothoracic amoebiasis can only be obtained by the identification of E.histolytica in the sputum, pleural or hepatic aspirates. Such confirmation was obtained more frequently in this study than in those

/previously ...

previously reported. Immediate examination of freshly obtained specimens by experienced workers was considered the likely explanation of the success in this respect.

PRIMARY PULMONARY AMOEBIASIS

The diagnosis of primary pulmonary amoebiasis, i.e. pulmonary amoebiasis of haematogenous origin without evidence of associated hepatic amoebiasis, may be extremely difficult. Manson-Bahr (1923) stated that in this variety of pulmonary amoebiasis radiological changes are of little help in the diagnosis and E.histolytica is seldom found in the sputum. He noted that the diagnosis in his patients was largely based on exclusion and the impressive response to a therapeutic trial of emetine. Others have suggested that a previous history of diarrhoea or dysentery or clinical or laboratory evidence of intestinal amoebiasis may be helpful in the diagnosis (Dorner and Friedlander, 1941).

As recently as 1956 Deaton and Garret stated that there had been no reported cases of embolic or haematogenous pulmonary amoebiasis in which E.histolytica had been identified in the sputum. This statement is incorrect as Putney and Baker (1938) and Lindsay et al.(1951) have reported patients with haematogenous amoebic lung abscess in whose sputa E.histolytica were identified.

There were no patients with primary pulmonary amoebiasis in this study and in a fairly extensive experience of pleuropulmonary

/amoebiasis ...

amoebiasis I have not encountered this presentation clinically or at necropsy at this hospital. The absence of associated hepatic amoebiasis, the wide range in character and situation of the pulmonary lesions and the infrequency with which E.histolytica are identified in the sputum appear to be the main factors contributing to the difficulty experienced in the diagnosis of this presentation.

THERAPEUTIC TRIALS IN THE DIAGNOSIS

Numerous workers have testified to the value of a therapeutic trial of emetine in the diagnosis of pleuropulmonary amoebiasis (Manson-Bahr, 1923; Spellberg and Zivin, 1948; Coirault et al., 1955; Sullivan and Bailey, 1951; Abdel-Hakim and Higazi, 1958). Recently Takaro and Bond (1958) stated that the diagnosis of pleuropulmonary amoebiasis in the absence of positively identifiable organisms in the sputum, aspirated or resected material rests on a combination of suggestive clinical and laboratory findings, plus a definite and dramatic response to emetine, chloroquine or both. They concluded that, when a presumptive diagnosis is made owing to the danger of these complications, amoebicidal treatment should be given promptly without waiting for laboratory confirmation.

In support of the above statements Arnott (1953) stated that he could recall several cases of amoebiasis in which the illness would have been prolonged indefinitely or the patient may have died

/if ...

if emetine had been withheld until the parasite had been found. Deschiens and Nelmette (1928) emphasised that the diagnosis of amoebiasis is too often made on flimsy evidence and stressed that a response to emetine is not necessarily diagnostic, a view shared by Mattei (1930) who suggested that emetine is of therapeutic value in pyogenic lung abscess.

Sullivan and Bailey (1951) stated that, although considerable diagnostic import may be attached to the response to emetine, the fallacies and disadvantages of the use of therapeutic agents as diagnostic measures must be appreciated. Wilmot (1962) expressed the opinion that caution is required in interpreting the results of therapeutic trials and stated that in some instances the apparent effect of emetine may be no more than coincidence.

Fortunately therapeutic trials of emetine and chloroquine were seldom required for purposes of diagnosis in my patients. In many instances the use of antibiotics prohibited diagnostic conclusions being formed on the basis of response to the tissue amoebicides. In those patients in whom the amoeba was not found the clinical features and investigations were usually such that the diagnosis could be made with reasonable confidence.

Therapeutic trials of emetine have probably saved many patients from the ill effects of undiagnosed pulmonary amoebic lesions. A clear-cut therapeutic response, particularly when other

/measures ...

measures have failed, supports the diagnosis. Such trials, however, are no substitute for clinical judgement and adequate investigation. The results obtained should always be interpreted with circumspection and should not blind the clinician to the possibility of error in his clinical diagnosis.

DIFFERENTIAL DIAGNOSIS

Conditions for which hepatotheracic amoebiasis may be mistaken are usually found at the right lung base and embrace a wide range of thoracic and subphrenic pathology (Berne, 1942). Infrequently the left lung base may be implicated (Dumade and Lavigneile, 1947; Ghosh, 1954) and rarely with primary or haematogenous pulmonary amoebiasis the lesion may be situated in any aspect of the lung fields (Chaudhuri and Chaudhuri, 1946; Chapman et al., 1948; Chatterjee and Sen Gupta, 1949).

Pulmonary tuberculosis appears to present the most frequent diagnostic problem, particularly basal tuberculosis (Dormer and Friedlander, 1941; Kilgore, 1951). Dell Aquila (1949) and others have reported examples of pulmonary amoebiasis masquerading as pulmonary tuberculosis (Bob, 1954; Heinemann, 1956), whilst others, reporting serous amoebic effusions, have noted that these may be mistaken for tuberculous effusions (Anagnostopoulos, 1940; Carruthers, 1947; Coirault et al., 1955). Oehsner and DeBakey (1936) stated that in their patients the basal situation of the pulmonary lesion and the tender hepatomegaly

/favoured ...

favoured amoebiasis as opposed to tuberculosis and others have noted that repeated failure to find acid-fast bacilli in the sputum is of value in excluding tuberculosis (Dormer and Friedlander, 1941).

Both Bockless (1950) and Chakravarti (1951) found that basal and viral pneumonias were the main source of confusion in the diagnosis of their patients, whilst others have noted that the basal pulmonary changes in amoebiasis may be mistaken for pneumonia (Rosencon and Bernard, 1928; Dormer and Friedlander, 1941; Manson-Bahr, 1945).

Pyogenic infections such as lung abscesses, bronchiectasis or empyema may present a problem in the differential diagnosis particularly when situated at the right base (Hughes and Westphal, 1947; Kossalka et al., 1949; Alarcon, 1954). Features such as the quantity and character of the sputum, the character of the pleural aspirate and the absence of pyogenic organisms, may be helpful in making the distinction (Ochsner and DeBakey, 1936).

Other thoracic conditions which have been mentioned in the differential diagnosis have been primary carcinoma of the lung (Manson-Bahr, 1945), multiple secondary carcinomatous deposits in the lungs (Cremier and Darbon, 1952), and pulmonary infarction (Alarcon, 1954).

Subphrenic presentations mentioned in the differential diagnosis of hepatothoracic amoebiasis have been, pyogenic liver

/or ...

or subphrenic abscess (Hughes and Westphal, 1947), cholecystitis, pylephlebitis (Manasem-Bahr, 1944), cirrhosis, infective hepatitis, carcinoma of the liver (Spellberg and Zivin, 1948; Conan et al., 1950) and hydatid disease (Alkan et al., 1961). Rarely with left basal hepatotheracic amoebiasis left upper quadrant or epigastric syndromes such as an acute peptic ulcer or pancreatitis require consideration (Alkan et al., 1961).

Typhoid and paratyphoid fever, malaria and brucellosis have been noted as systemic diseases with which hepatotheracic amoebiasis may be confused (Alarcon, 1954; Webster, 1960).

Pulmonary conditions presenting with clinical and radiological changes at the right base such as pneumonia, pyogenic lung abscess, bronchiectasis, carcinoma with atelectasis and pulmonary infarction were those most frequently considered in the differential diagnosis in this study. Classical apical parenchymal tuberculosis was seldom a problem in the differential diagnosis, but basal pulmonary tuberculosis had to be excluded.

Foremost in the differential diagnosis of patients in this study presenting with pleural effusions was tuberculous empyema or effusion, whilst others were pyogenic empyema, pneumonia with effusion and carcinoma with effusion.

Subphrenic presentations with diaphragmatic involvement required exclusion in my patients. These were essentially similar

/to ...

to those mentioned above in the review of the literature. Unfortunately there is a tendency for the anatomical barrier formed by the diaphragm to limit one's diagnostic reasoning. Because of this, subphrenic conditions such as subphrenic abscess, carcinoma of the liver, hydatid disease and pancreatitis, all of which can present with signs in the chest, may be overlooked when considering the differential diagnosis of unexplained changes at the base of the lung. In some instances disseminated disease such as septicaemia with pylephlebitis and pulmonary involvement, disseminated tuberculosis or carcinomatosis presented problems in the differential diagnosis.

CONCLUSIONS

The diagnosis of hepatotheracic amoebiasis is suggested in many instances by a fairly characteristic clinical presentation.

Hepatopulmonary amoebiasis should be suspected when symptoms and signs referable to the right lower chest are associated with a tender hepatomegaly or guarding and tenderness in the right upper quadrant of the abdomen. The production of large quantities of typical anchovy sauce sputum is highly suggestive of the diagnosis. Radiologically, the finding of an elevated or immobile right diaphragm with or without parenchymal changes at the right lung base supports the diagnosis which is proved by the identification of *E.histolytica* in the sputum.

The combination of a pleural effusion and a tender enlarged liver or guarding and tenderness in the right upper quadrant of the abdomen should suggest the possibility of hepatopleural amoebiasis. When the pleural pus aspirated from patients with the above combination is sterile on culture for bacteria and acid-fast bacilli the diagnosis should be suspected. The aspiration of typical anchovy sauce pus from the pleural cavity is highly suggestive of an amoebic empyema whilst the identification of E.histolytica in the pleural pus proves the diagnosis.

In any patient with pleural or pulmonary lesions, particularly when these are situated at the lung base, the diagnosis of hepatothoracic amoebiasis should be suspected if there are findings compatible with an amoebic liver abscess of the right or left lobe of the liver. Aspiration of pus from the liver proves the existence of such an abscess, and when this pus is of an anchovy sauce colour or sterile on culture an amoebic origin is likely, whilst the finding of E.histolytica in the pus proves the diagnosis.

Pleuropulmonary amoebiasis should be suspected in all patients with undiagnosed pleural or pulmonary lesions. Evidence of intestinal amoebiasis when present may suggest an amoebic origin of the above lesions but absence of such evidence in no way excludes this possibility. In such presentations a clear cut response to a therapeutic trial of emetine and chloroquine supports the diagnosis to some extent.

The differential diagnosis of pleuropulmonary amoebiasis includes a wide range of conditions involving the base of the right chest and/or the right upper quadrant of the abdomen. Uncommonly lesions at the base of the left lung and/or in the epigastrium or left upper quadrant are implicated. Rarely pathology in any aspect of the lung fields may require consideration.

THE MANAGEMENT OF PLEUROPULMONARY AMOEBIASIS

There are three important aspects to the management of pleuropulmonary amoebiasis. The first of these concerns chemotherapy, the second, drainage procedures and the third, the indications for major surgery.

CHEMOTHERAPY

Emetine hydrochloride: For almost 40 years after the demonstration of its amoebicidal action by Vedder and successful clinical use by Rogers in 1912, the ipecacuanha alkaloid, emetine, remained the only available effective preparation for the treatment of extra-intestinal amoebiasis (Rogers, 1912; Vedder, 1914). Following Roger's lead Chaufford in 1913 successfully used emetine in a patient with a hepatobronchial fistula and since this time numerous workers have testified to its efficacy and have been impressed by the rapid, sometimes dramatic, response obtained in the treatment of pleuropulmonary amoebiasis (Miginiac, 1922; Manson-Bahr, 1923; Oehsner and DeBakey, 1936,1939,1942; Blanc and Siguier, 1946; Bookless, 1950; Abdel-Hakim and Higazi, 1958). Some, in addition to giving the drug intramuscularly, have injected solutions locally into the liver abscess or pleural space (McHardy, 1951; Abdel-Hakim and Higazi, 1958).

There are those who maintain that the effect of emetine is specific, being restricted to conditions such as amoebiasis and paragoniniasis (Rogers, 1922; Sullivan and Bailey, 1951), and that

/because ...

because of this therapeutic trials are of value in the diagnosis of thoracic amoebiasis. Other parasitic disorders in which emetine is of known value are bilharziasis (Manson-Bahr, 1945) and fascioliasis (Kouri and Arenas, 1932). It has also been used in the treatment of non-parasitic conditions such as pyogenic lung abscess (Mattei, 1930).

Emetine is a cytoplasmic poison and its toxic manifestations have been well documented (Brown, 1935; Dack and Meleshek, 1947; Goodman and Gilman, 1955; Wilmot, 1962). The most serious of these are its effects on the myocardium which may manifest by tachycardia, arrhythmias, alterations in blood pressure and electrocardiographic changes. The incidence of toxic reactions reported varies widely from 2 to 3 per cent (Manson-Bahr, 1941; Brown, 1935) to 91 per cent (Klatskin and Friedman, 1948). My experience of the toxic reactions of emetine in several hundred patients suggests that the frequency quoted by Manson-Bahr and Brown is the more realistic. In Rail's experience toxic effects of emetine were uncommon and Adams expressed the opinion that the dangers of emetine as a myocardial toxin have been grossly exaggerated (Rail, 1947; Adams, 1956).

Fatalities have been attributed to the use of emetine (Levy and Mountree, 1916; Leibly, 1930; Bren and Kenwaler, 1955) but Wilmot (1962) has pointed out that in some of these patients the drug was given in unacceptable doses and in others, associated disease processes probably contributed to the outcome.

In 1952 Adams stated that emetine remains the most effective drug available for the clinical control of amoebiasis and that there is no other drug which is its equal in this respect. I believe this statement to be as true now as it was 10 years ago. With the exception of a few in whom dehydroemetine was substituted, all patients in this study received emetine. This drug was considered the cornerstone of treatment in my patients and serious toxic manifestations resulting from its administration were rare (case 41). A troublesome but not serious finding was that many patients experienced pain and tenderness at the site of injection, whilst in a few "emetine abscesses" developed.

Recently the synthetic analogue of emetine, dehydroemetine, has been used in the treatment of hepatic amoebiasis with good results and there have been claims that the preparation is equally effective, but less toxic than emetine (Gonzalez De Cossio, 1960; Ortiz De Montellano, 1961; Blanc et al., 1961; Rosenstiel, 1961). Wilmot et al. (1963) have shown that a dehydroemetine-chloroquine combination is equally as good as an emetine-chloroquine combination in the treatment of amoebic liver abscess. In the few patients in this study in whom dehydroemetine was substituted for emetine a satisfactory response resulted in each instance.

Chloroquine (diphosphate or sulphate): In 1948 Conan et al. published a report on the successful treatment of hepatic amoebiasis with the proteosuccide chloroquine. These workers subsequently reported its

/efficacy ...

efficacy in the treatment of pulmonary amoebiasis and stated that the drug attained high levels of concentration in pulmonary as well as hepatic tissue (Conan et al., 1950).

Others who have found chloroquine of value in the treatment of thoracic amoebiasis have been Basnuevo et al. (1950), Chakravarti, (1951,1952,1953), Takaro and Bond (1958) and Cook-Sup So (1959). Indeed some have been so impressed with its effectiveness and relative lack of toxicity, that there has been a tendency to use it in preference to emetine (Basnuevo and Estarli, 1949; Basnuevo et al., 1950; Gambardella and De Michele, 1951; Zavala and Hamilton, 1952; Fry, 1959; Webster, 1956,1960). This tendency is unfortunate when it is remembered that Wilmot et al. (1958,1959) have shown that, in the treatment of proved amoebic liver abscess, the use of chloroquine alone resulted in a relapse rate of over 25 per cent, while with the usual two courses of emetine this figure was nil. Harinasuta (1951) has also noted a higher relapse rate with chloroquine as opposed to emetine and Alarcon (1954) reported a patient with pulmonary amoebiasis who failed to respond to chloroquine but showed the usual satisfactory response to emetine.

In addition to the oral administration of chloroquine Basnuevo et al. (1951) recommended the injection of a 2 per cent solution into abscess cavities as a routine in treatment. Chloroquine given orally is highly concentrated in hepatic and pulmonary tissues (Conan et al.,

/1950...

1950) and, because of this, I consider its local injection into abscess cavities unnecessary.

Emetine and Chloroquine: Patients have been reported with pulmonary amoebiasis who have not responded to treatment with emetine (Gamberdella and de Michele, 1951) and there have been those in whom chloroquine was ineffective (Alareem, 1954). Wilmot et al. (1958,1959) noted that the unfortunate tendency to use chloroquine alone in the treatment of hepatic amoebiasis has already brought its toll of tragedies. They stated that the chances of cure are greatest when a combination of emetine and chloroquine are employed in treatment. Others who have used a combination of emetine and chloroquine in pleuropulmonary amoebiasis have been Lamont and Pooler (1958) and Singh and Jelly (1962).

All patients in this study were treated with a combination of emetine and chloroquine. Chloroquine was never used alone and, in my opinion, unless some definite contraindication to emetine exists, it should never be used alone in the treatment of pleuropulmonary amoebiasis. Nausea and vomiting were the toxic manifestations encountered with chloroquine but these seldom necessitated discontinuing treatment. Local injection of solutions of chloroquine and/or emetine into the pleural space or liver abscesses was considered unnecessary in my patients as it has been shown that these drugs reach high levels of concentration in both hepatic and pulmonary tissues (Farmer and Cottrill, 1949; Conan et al., 1950). In my opinion the rapid response

/in ...

in most instances and the low mortality rate in this study are attributable in part to the use of these two potent tissue amoebicides.

Antibiotics: Antibiotics have been shown to be of value in the treatment of intestinal amoebiasis (Hargreaves, 1945; Armstrong et al., 1949,1950), and the tetracyclines have proved to be the best of those tried to date (Elsdon-Dew et al., 1952; Wilmot, 1955).

The tetracyclines, chloramphenicol and erythromycin have been amongst the antibiotics tried in the treatment of hepatic amoebiasis (Wilmot et al., 1952,1958; McHardy and Frye, 1954). The results obtained indicate that these drugs are inferior to the recognised tissue amoebicides in the treatment of hepatic and other varieties of extra-intestinal amoebiasis (Abdel-Hakim and Higazi, 1958). "Their use alone can only lead to tragedies" (Wilmot, 1962).

When secondary bacterial invasion of an amoebic liver abscess or amoebic empyema occurs local and systemic antibiotics should be administered (Wilmot, 1962). Basmuevo et al. (1951) included local and systemic antibiotics in the therapy of their patient in whom an amoebic liver abscess had ruptured into the lung.

Antibiotics were probably administered more frequently than was necessary in this study. This was because the decision as to when they were required was not always under my control. It has been previously noted that parenteral and local antibiotics should be used when there is evidence of secondary bacterial invasion. Wilmot (1962) has suggested

/that ...

that the use of an antibiotic is probably wise in an amoebic lung abscess as bacteria have access to the cavity and may delay or prevent healing. In my experience, provided drainage is adequate, an amoebic lung abscess responds more rapidly to emetine and chloroquine than its pyogenic counterpart to antibiotics. For this reason I believe that antibiotics are not required in the treatment of most patients with an amoebic lung abscess. I consider that the indications for antibiotics in pleuropulmonary amoebiasis should be as follows:

(i) In instances in which the amoebic pathology is complicated by secondary bacterial invasion or when the clinical features and investigations suggest this complication.

(ii) In patients with concomitant amoebic dysentery.

(iii) When the diagnosis is in doubt and pyogenic pulmonary disease is suspected.

Staphylococci and gram-negative bacteria resistant to a variety of antibiotics are frequently cultured from the pus of secondarily infected amoebic liver abscesses at this hospital. As it is impossible to predict in advance the antibiotics to which these organisms will be sensitive, I feel it is questionable whether antibiotics should be used prophylactically to prevent bacterial invasion of amoebic liver or pleuropulmonary lesions.

Miscellaneous Preparations: Other drugs used in the treatment of pleuropulmonary amoebiasis have been ipecacuanha (Hansen-Bahr, 1923), treparsol (Grasset and Fourquier, 1928), carbasone (Dermer and Friedlander, 1941), cemesaine (Soulage, 1949) and mepacrine (Radke, 1951,1952; Abd-El-Ghaffar and Abd-El-Ghaffar, 1955). Since the advent of its alkaloid emetine, ipecacuanha is no longer required as a tissue amoebicide and when emetine and chloroquine are used in combination there is no necessity to use the other preparations mentioned above in the treatment of pleuropulmonary amoebiasis.

DRAINAGE PROCEDURES

Amoebic Liver Abscess - Closed Drainage: The closed or aspiration method of evacuating liver pus was advocated by Annesley as long ago as 1828. Maclean (1871) observed a better prognosis in those who developed a hepatobronchial fistula and coughed up the abscess contents than in those subjected to open drainage. On this basis, he attempted closed drainage with repeated aspiration and reported an appreciable reduction in mortality with this method.

Having shown that the majority of amoebic liver abscesses are bacteriologically sterile, Rogers in 1902 proposed that secondary bacterial infection was the principal cause of death following surgical incision and open drainage. Against considerable opposition he advocated closed drainage with repeated aspiration and the injection of quinidine into the abscess cavity (Rogers, 1922). In 1912, a milestone in the

/treatment ...

treatment of amoebiasis was reached when Rogers added injections of the soluble salts of emetine to his aspiration routine in the management of an amoebic liver abscess.

The emetine plus aspiration or closed drainage sequence, first proposed by Rogers (1912) has been shown repeatedly to be the most satisfactory regime for the management of an amoebic liver abscess (Thurston, 1914; Manson-Bahr, 1931; Ochsner and DeBakey, 1939,1943,1951; Klatskin, 1946; Lamont and Peeler, 1958).

The treatment of pleuropulmonary amoebiasis according to Ochsner and DeBakey (1936) consists of the administration of emetine and aspiration of those liver abscesses not sufficiently evacuated through a bronchus. They stated that open drainage should never be done except in those cases with secondary infection. Others who have expressed agreement with the above principles of management have been Chakravarti, 1951,1952,1953; Takaro and Bond, 1958; Abdel-Hakim and Higazi, 1958; Wilmot, 1962).

Open Drainage: In the early part of this century surgical incision with open drainage was common practice in the treatment of an amoebic liver abscess. There were those who preferred a transthoracic approach (Ludlow, 1917; Petridis, 1924), others a laparotomy (Cignozzi, 1925) whilst others were influenced by the clinical presentation in their choice of an abdominal or thoracic incision (Lacaze and Helmotte, 1928). In addition to incisional drainage some curetted

/the ...

the abscess cavity (Fenton, 1909), some packed it with gauze (Ludlow, 1917) and others injected emetine into its substance (Job and Spick, 1917).

A few have reported excellent results (Fenton, 1909; Lacaze and Malmotte, 1928) but the majority experienced a high mortality following surgical incision and open drainage of amoebic liver abscess (Sambon, 1911; Rogers, 1922; Petridis, 1924; Berne, 1942). Chatterji (1927) employed surgical incision with open drainage in 67 patients, with a mortality of 27 per cent and closed drainage via aspiration in 186 patients, with a mortality of only 6 per cent.

Although emetine administration with aspiration drainage of an amoebic liver abscess is now accepted as the treatment of choice, indications for surgical drainage still exist and when present should not be ignored (Wilmot, 1962). Mansen-Bahr and others have stated that indications for surgery may be thick pus prohibiting aspiration, secondary infection or rupture of the liver abscess into one of the adjacent serous cavities (Chatterji, 1927; Mansen-Bahr, 1944; Rogers and Megaw, 1946). Ochsner and DeBakey (1936) advised strongly against open drainage in the management of pleuropulmonary amoebiasis. In their collected series a mortality of 48.2 per cent when surgical drainage was used alone was reduced to 16.6 per cent when emetine was included with surgical incision, whilst in those receiving emetine alone the mortality was only 5.4 per cent. These workers considered that open drainage should never be done except in those cases with secondary infection. More recently Wilmot (1962)

/noted ...

noted that a laparotomy may be required to confirm or disprove the diagnosis and drain a liver abscess when other measures have failed. He felt that surgical drainage should be considered when the response is unsatisfactory following adequate drug treatment and repeated aspiration, or when aspiration of a left lobe liver abscess has been inadequate to avoid the danger of intrapericardial rupture.

In this study closed drainage with needle aspiration was employed to evacuate pus from amoebic liver abscesses. In some patients aspiration was unsuccessful, in others it was not required as adequate drainage resulted through the hepatobronchial communication. It was found that if a patient coughed up more than 200 ml. of pus daily through a hepatobronchial fistula, he was not likely to require needle aspiration to drain his liver abscess.

Excluding the above exception, the indications for liver aspiration in my patients were essentially similar to those proposed by Wilmot (1962).

(i) Localised swelling or bulging of the right lower thoracic cage.

(ii) Signs at the right base suggesting elevation of the right diaphragm or radiological evidence of the same.

(iii) Marked localised tenderness or oedema.

(iv) Lack of response to specific treatment shown by persistence of constitutional symptoms after 5 to 7 days of treatment or continued hepatic enlargement and tenderness.

Complications of liver aspiration such as haemorrhage (Teullee and Huard, 1934), secondary bacterial contamination (Ochsner and DeBakey, 1936) and external fistula (Wilmot, 1962) were not encountered.

Amongst my 50 patients with pleuropulmonary amoebiasis there was only one instance (case 31) when surgical evacuation of the responsible liver abscess was required. Indications for surgical incision and drainage of a liver abscess in this study were similar to those proposed by Wilmot (1962).

Amoebic Empyema: In 1920 Proust and Ramond reported a patient suffering from an amoebic empyema who was cured with repeated aspirations and emetine. They rejected open drainage of the empyema as the method of choice and recommended medical treatment with repeated aspirations.

Reviewing 28 cases of amoebic empyema, 24 of whom died, Verges and Hermenjat-Gerin (1932) recommended immediate thoracotomy with aspiration of the pus, debridement of the diaphragmatic opening and drainage of the liver abscess contents. If the pus removed was free of bacteria they repaired the opening in the diaphragm; if however bacteria were identified, the abscess cavity was marsupialised and a drain left *in situ*. Others who have recommended surgical incision with open drainage to evacuate an amoebic empyema have been (Huard and Meyer-May, 1936; Manson-Bahr, 1944; Rogers and Megaw, 1946).

Ochsner and DeBakey (1936) felt that the high mortality in their personal and collected cases of amoebic empyema resulted because emetine was used in few instances and open drainage frequently employed.

Based on their personal series of 5 cases and a collected series of 27 cases these workers recommended repeated aspiration as the method of choice for drainage and considered that open drainage was only required when secondary infection of the empyema resulted. Shaw (1949) considered that surgical drainage is required in amoebic empyemas not responding to repeated aspiration, or in those which are secondarily infected.

Eight out of 9 of the patients with amoebic empyema reported by Lamont and Peeler (1958) recovered following closed drainage of the empyema by repeated aspiration and treatment with emetine and chloroquine. Wilmot (1962) recommended repeated aspiration with as much pus as possible being removed at each aspiration. He stated that surgical drainage is unnecessary unless the volume of pus withdrawn at successive aspirations is not diminishing, the patient's condition is deteriorating, or secondary infection with bacteria occurs.

There are few workers with an extensive experience in the management of amoebic empyema. From the above review it is apparent that there is no general agreement as to which is the best method of removing the pus. In this study 9 of the 13 patients in whom needle aspiration was used to drain the empyema made a complete recovery. Of the remaining 4, one patient (case 44) was left with considerable pleural thickening and 2 patients (cases 37 and 41) died. The reasons why closed drainage by needle aspiration was used in this study are twofold. Firstly it has been my experience that the chances of secondary bacterial

/contamination ...

contamination are much less likely with this method and secondly, ill patients are spared the additional insult of a general anaesthetic and rib resection required to introduce a tube drain of sufficient calibre for open drainage. It may be argued that there is relatively little trauma associated with the introduction of an intercostal tube drain through a cannula but I have found this method of open drainage unsatisfactory. The results with closed drainage by needle aspiration in my patients have certainly justified its use. A factor which contributed to the success achieved with this form of drainage was that as much pus as possible was removed at each aspiration.

In my patients closed drainage was considered to have failed when the pus aspirated was too thick to be removed in quantity or the quantity removed at successive aspiration was not diminishing. In such instances, which were rare, open drainage was employed. Secondary bacterial invasion was not considered an indication for thoracotomy and drainage unless the infected pus could not be adequately removed by needle aspiration.

As mentioned previously injection or lavage of the empyema cavity with emetine and chloroquine was considered unnecessary in my patients. In the rare instances in which secondary infection with bacteria occurred solutions of the appropriate antibiotics were injected locally.

Postural Drainage: In 1912 McKechnie demonstrated the importance of postural drainage in the management of pulmonary amoebiasis. He described a patient with a hepatobronchial fistula who had coughed up pus for 5 years who responded satisfactorily to postural drainage as the only treatment. Rogers (1922) considered extension of a liver abscess through the diaphragm with drainage through a bronchus as "nature's" method of drainage and claimed a recovery rate of more than 50 per cent following this event. Higginas (1922) noted that when a hepatobronchial fistula is providing adequate drainage of a liver abscess, aspiration of the abscess is not required. In the rare instances when there is no response to postural drainage and drug therapy, aspiration of the abscess should be tried and, if unsuccessful, surgical drainage may be necessary. More recently Wilmot (1962) included postural drainage in his recommended management of amobic lung abscesses.

The value of postural drainage in the management of pulmonary amoebiasis has not been sufficiently stressed in the literature. I find it surprising that, in a number of fairly comprehensive articles on pulmonary amoebiasis, no mention has been made of this aspect of treatment (Cohnmer and DeBakey, 1936; Takaro and Bond, 1958; Abdel-Hakim and Higazi, 1958). Perhaps it was considered too elementary to deserve attention. In my patients with hepatobronchial fistulae and lung abscesses adequate postural drainage was considered to be amongst the most important aspects of management as in the majority large

/quantities ...

quantities of pus were drained by this method.

INDICATIONS FOR SURGERY

In 1949 Kossalka et al. advised that the thoracic complications of an amoebic liver abscess should be handled surgically and proposed such measures as thoracotomy with resection of involved pulmonary tissue. Opposing the above view are authors such as Ochsner and DeBakey (1936), Takaro and Bond (1958) and Wilmot (1962) who advocate conservative measures in the management of pleuropulmonary amoebiasis. The indications for surgical incision and drainage in the acute stage have been discussed. It remains to review and discuss the indications for surgery in the chronic stage of the illness.

Among the residual lesions of pulmonary amoebiasis which may require surgery Shaw (1949) mentioned persistent hepatobronchial fistulae and areas of pulmonary fibrosis resulting from an amoebic lung abscess which are producing symptoms. Brandon et al. (1957) discussed Shaw's proposals and included with these decortication of the lung in patients with residual pleural thickening and chronic amoebic empyema. Others have stated that failure of the lung to re-expand is an indication for thoracotomy (Ayas and Aranus, 1950).

Takaro and Bond (1958) stated that surgery may be avoided in many patients if the underlying hepatic abscess is treated before it involves the thorax which is a somewhat elementary but very true statement. They felt that surgery should be withheld unless specific

/indications ...

indications arise, and even then, should be used only after adequate amoebicidal drug therapy has been given. They considered indications for surgery to be as follows:

(i) secondary infection of a liver or lung abscess, or of empyema;

(ii) persisting hepatobronchial fistula;

(iii) permanently damaged lung;

(iv) drainage or resection of an abscess of unknown etiology;

(v) failure to respond to conservative management.

Recently Basu Chaudhuri et al. (1960) noted that occasionally an abscess cavity will persist or bronchiectasis may develop in a patient with pulmonary amoebiasis and when these residual lesions are producing symptoms surgical excision of the affected segment should be undertaken. Before embarking on resection of the residua of pulmonary or pleural amoebiasis Wilmot (1962) advised that a long period of observation is essential as improvement may continue for many months after the completion of specific treatment.

Early diagnosis with administration of emetine and chloroquine with adequate drainage by needle aspiration when required were undoubtedly the main reasons why major surgery was seldom required in my patients. There were no patients with persistent hepatobronchial fistulae and

/consequently ...

consequently surgery for this eventuality was not required. Four patients were shown to have residual right basal bronchiectasis by bronchography but as these lesions were not producing symptoms surgery was not advised. One patient with residual fibrosis and probable bronchiectasis (case 39) refused to consider surgery. There was only one patient (case 42) with extensive residual pleural thickening who might have benefited from decortication. He refused to consider surgery.

The main reason why these patients refused to consider surgery was that their residual lesions caused them little or no disability and I would have done the same in their position. It may be concluded then that major surgery is seldom required in the management of pleuropulmonary amoebiasis and should only be contemplated if the residual lesions are producing symptoms or disability which will definitely be improved by surgical measures.

CONCLUSIONS

Emetine hydrochloride is the most effective single preparation available for the treatment of pleuropulmonary amoebiasis and unless a definite contraindication exists it should be used in all patients with this condition. Emetine is a myocardial toxin but its dangers in this respect have been exaggerated. When used correctly and the dose adjusted in the young, in the aged and in wasted patients serious side effects are rare.

The pulmocoxide chloroquine (phosphate or sulphate) is of value in the treatment of pleuropulmonary amoebiasis and should be used in combination with emetine. It has been shown that this drug is not as effective as the usual two courses of emetine in the treatment of amoebic liver abscess and it should therefore not be used alone in the treatment of the more serious thoracic complication.

A small number of patients with pleuropulmonary amoebiasis will require more than one course of emetine and chloroquine. In such circumstances the recognised time interval of 10 days to 2 weeks should be observed before giving the second course of emetine.

Antibiotics may be required in addition to emetine and chloroquine in the treatment of pleuropulmonary amoebiasis. They should not be used alone. Indications for their use are, evidence of secondary bacterial invasion, concomitant amoebic dysentery and when the diagnosis is in doubt and pyogenic pulmonary disease suspected.

Adequate drainage of the responsible liver abscess is essential in pleuropulmonary amoebiasis. This may occur when the pus is coughed up through a hepatobronchial fistula or when pleural aspiration drains the pus through a hepatopleural fistula. When the liver abscess is of sufficient size to indicate its presence by clinical features such as localised tenderness, swelling and oedema, or an elevated right diaphragm, needle aspiration should be attempted. Because of the danger of secondary bacterial invasion needle aspiration as opposed to open drainage procedures should be used to remove the pus from an amoebic liver abscess. Open

/drainage ...

drainage may be required in pleuropulmonary amoebiasis when drainage by needle aspiration is inadequate.

Adequate drainage of an amoebic empyema is essential. Because of the danger of secondary bacterial invasion needle aspiration rather than open drainage procedures should be used. As much pus as possible should be removed at each aspiration and aspirations should be repeated at 48 to 72 hour intervals until no further quantities of pus can be obtained. Open drainage of an amoebic empyema may be required when the pus is too thick to be removed by needle aspiration, when the quantity of pus removed at successive aspirations is not diminishing or there is evidence that the pus is loculated. If secondary bacterial infection of an amoebic empyema occurs solutions of the appropriate antibiotic should be injected into the pleural cavity. If the infected pus cannot be adequately drained by needle aspiration open drainage should be instituted.

Postural drainage is a necessary and valuable aspect in the treatment of hepatobronchial fistula or amoebic lung abscess.

When pleuropulmonary amoebiasis is managed as advised above and there is no undue delay in commencing treatment major surgical procedures such as decortication or the resection of pulmonary tissue are seldom required.

PROGNOSIS IN PLEUROPULMONARY AMOEBIASIS

In the series of 28 patients with amoebic empyema collected from the literature by Verges and Hermenjat-Serin (1932), 24 (86 per cent) died. It was claimed that the high mortality resulted from inadequate treatment; immediate surgical incision, open drainage and treatment with emetine and arsenicals were recommended.

In their collected series of 153 patients with pleuropulmonary amoebiasis Ochsner and DeBakey (1936) reported a mortality of 41.1 per cent. They stated, "the prognosis depends probably more upon the type of therapy than upon the type of lesion." In their patients the recovery rate in those who were treated without emetine was 43.9 per cent, whereas in those who received emetine it was 91.8 per cent. It was considered that the use of open drainage as opposed to closed drainage was a further important factor adversely affecting the prognosis. They suggested that had open operation been used less frequently and emetine more frequently, the mortality rate would have been much lower.

Table 13 shows the recovery rates reported in pleuropulmonary amoebiasis in recent years. A glance at this table indicates that the prediction of Ochsner and DeBakey appears to have been substantiated. The 100 per cent survival rate reported by Chakravarti (1951,1952,1953) and Abdel-Hakim and Higasi (1958) should be accepted with reservation as no mention was made of how their patients were selected. There was

/no ...

TABLE 13

RECOVERY RATES REPORTED IN PLEUROPULMONARY AMOEBIASIS IN RECENT YEARS

Author	Year	Type of Lesion	No. of Cases	Recovery
Chakravarti	1951-53	Pulmonary	26	26 (100%)
Lamont and Pooler	1958	Pulmonary	17	16 (94%)
		Pleural (Empyema)	9	8 (89%)
Abdel-Hakim and Higazi	1958	Pulmonary	20	20 (100%)
		Pleural (3 Empyemas)	8	8 (100%)
Tahara and Bond (review)	1958	Pulmonary	166	131 (79%)
		Pleural (Empyema or Effusion)	62	52 (84%)

no selection in the group reported by Lamont and Peeler (1958) whose patients were encountered among 250 with amoebic liver abscess. In the figures taken from the review by Takaro and Bond (1958), 52 patients in whom the character of the lesion remained unknown have been omitted. When the above results are compared with those reported by Verges and Marmonjat-Gerin (1932) and Ochsner and DeBakey (1936) it can be seen that there has been a noticeable improvement in recent years. The two factors which have probably contributed most to this improvement in recovery rate, are firstly, a greater number of patients in recent times have been adequately treated with emetine and or chloroquine and secondly, closed drainage by needle aspiration is more frequently used than open drainage by surgical incision.

In this study there were 2 deaths (7 per cent) in the pulmonary group and 4 (20 per cent) in the pleural group with an over-all mortality of 12 per cent. The highest mortality occurred in the 15 patients with amoebic empyema of whom 4 (27 per cent) died. In one of these (case 38) death was due to an associated amoebic brain abscess and in another (case 43) it occurred shortly after an acute intra-pleural rupture of a liver abscess. In one patient in the pulmonary group (case 6) delay in diagnosis undoubtedly contributed to the fatal outcome.

In addition to the higher mortality it was noticed that morbidity was greater and response to treatment slower in those with amoebic empyemas than in those with pulmonary lesions. Factors which

/appeared ...

appeared to influence the response to treatment in my patients were the age and general condition, the duration of symptoms and the character of the lesion.

The recovery rate of 88 per cent in this study compares favourably with that of Lamont and Pooler (1958) and Takaro and Bond (1958) recorded in table 13. In my opinion the factors responsible for the good prognosis in my patients were, early diagnosis, early treatment with emetine and chloroquine and adequate drainage of pus by needle aspiration rather than surgical incision.

It is impossible to ascertain whether the use of both emetine and chloroquine in my patients as opposed to emetine or chloroquine alone influenced the prognosis. Judging from the results reported by Wilmot et al. (1958,1959) in the treatment of uncomplicated amoebic liver abscesses with this combination, it is likely that their combined use reduced morbidity.

In my experience secondary bacterial invasion worsens the prognosis in pleuropulmonary amoebiasis. Fortunately there were too few patients in this study with this complication to assess its influence on the prognosis.

Unfortunately the attendance at follow-up clinic (28 per cent) was too poor to make a definite statement on the long term prognosis in pleuropulmonary amoebiasis. Judging from those patients who continued to attend, however, and my past experience of this condition, when it is managed along the lines proposed in this study, the long term

/prognosis ...

prognosis is excellent.

CONCLUSIONS

A good prognosis in pleuropulmonary amoebiasis is dependent upon early diagnosis followed by treatment with emetine and chloroquine, and adequate drainage of pus by conservative methods of needle aspiration and postural drainage.

Mortality in pleuropulmonary amoebiasis appears to be greater in patients with amoebic empyema than in those with pulmonary lesions.

PART II - PERICARDIAL AMOEBIASIS

THE FINDINGS IN 15 PATIENTS WITH AMOEBIC PERICARDITIS

CLINICAL FINDINGS (Cases 51 - 65)

Age: This group comprised 15 African males whose ages ranged from 17 to 49 years with an average age of 32 years.

Duration of History: The shortest history was of 3 days (case 52) and the longest of one year (case 62) with an average duration for the group of 10 weeks. In 5 patients symptoms had been present for a month or less.

Main Complaints: The main clinical features have been recorded in table 14. Major complaints were of low retrosternal and or epigastric pain (15 patients). Eleven complained of low retrosternal pain, 9 of epigastric pain, and in 4 pain was experienced in both sites. There was radiation of the pain to the left shoulder tip in 5 (cases 52,53, 54,55 and 64) instances. Four patients complained of associated pleuritic pain in the left lower chest (cases 52,53,61 and 64) and one (case 62) experienced pleuritic pain in the right lower chest.

Eleven complained of dyspnoea on exertion, 12 of a non-productive cough and 3 (cases 60,61 and 64) of swelling of the feet.

One patient (case 59) admitted to a past history of dysentery and one (case 58) had been treated in this hospital 6 months prior to

/admission ...

TABLE 14

CLINICAL FEATURES OF 15 PATIENTS WITH ANOMIC PERICARDITIS

Symptoms	Patients	Per Cent
Pain low retrosternal or epigastric	15	100
Pain retrosternal	11	73
Pain epigastric	9	60
Pain left shoulder tip	5	33
Dyspnoea on exertion	11	73
Cough non-productive	12	80
Swelling of the feet	3	20
Past history of dysentery	1	7
Signs		
Pyrexia on admission	9	60
Pale mucosa	9	60
Evidence of recent weight loss	8	53
Ankle or sacral oedema	5	33
Tachycardia of 120 per min. or more	10	67
Small volume to the peripheral pulse	10	67
Pulsus paradoxus	10	67
Systolic blood pressure of 100 mm. Hg or less	8	53
Pulse pressure of 30 mm. of Hg or less	8	53
Raised jugular venous pressure	13	87
Increased cardiac dullness	10	67
Gallop rhythm	12	80
Pericardial friction rub	8	53
Tender hepatomegaly	15	100
Tenderness maximal in the epigastrium	9	60
Localised epigastric swelling	2	13

admission for an amoebic liver abscess of the right lobe.

General Examination: Nine patients were pyrexial on admission and 6 subsequent to admission. In 13 the temperature remained below 101°F and pyrexia of a low grade non-specific type was usual.

Pallor of the mucous membranes was noted in 9 patients and ankle and or sacral oedema in 5 (cases 52,56,60,61 and 64). Eight showed evidence of recent weight loss and 2 (cases 59 and 65) were emaciated.

Local Examination: On admission 11 patients presented with signs suggestive of pericarditis or pericardial effusion. Ten had a pulse rate of 120 or more per minute and in 10 there was a small volume to the peripheral pulse and a palpable pulsus paradoxus.

A systolic blood pressure of 100 mm. of Hg or less was found in 8 patients and in 8 the pulse pressure was 30 mm. of Hg or less. The jugular venous pressure was raised in 13. The area of cardiac dullness was increased in 10 and the apex beat was nearly always impalpable. A gallop rhythm was present in 12 and a pericardial friction rub heard in 8.

Two patients (cases 54 and 56) who did not present with signs of pericardial involvement subsequently developed these signs. In one (case 54) signs of pericarditis were found on the 2nd day and in the other (case 65) the signs of a pericardial effusion suddenly appeared on the 4th day.

The 2 remaining patients (cases 57 and 58) were considered to be suffering from cardiac failure and the diagnosis of pericarditis was not established until necropsy.

Signs suggestive of a raised left diaphragm or left basal pleural effusion were found in 3 patients (cases 55,61 and 65) and in 2 (cases 56 and 63) there were signs suggestive of a right basal effusion or elevated right diaphragm.

Epigastric guarding and tenderness with a tender hepatomegaly were found in all 15 patients on admission. In 9 tenderness was maximal in the epigastrium. A localised epigastric swelling was visible in 2 patients (cases 51 and 60).

On the basis of the clinical presentation, investigations such as pericardial aspiration, the response to treatment and subsequent progress, patients with amoebic pericarditis were classified as pre- or non-suppurative (cases 51 - 55) and suppurative presentations (cases 56 - 65). It was only possible in one instance on the basis of the clinical presentation to postulate the existence of presuppurative as opposed to a suppurative pericarditis. In this patient (case 54) the only evidence of pericarditis was a transient pericardial friction rub and signs of tamponade were absent. The other 4 patients (cases 51,52, 53 and 55) in the presuppurative group each presented with signs of cardiac tamponade and in these the diagnosis of presuppurative pericarditis was based on findings which will be recorded in due course.

Of the 10 patients (cases 56 - 65) in the suppurative group

/7 ...

7 presented with signs of severe cardiac tamponade on admission and a further patient (case 65) suddenly developed signs of cardiac tamponade on the 4th day. In the 2 remaining patients in this group (cases 57 and 58) the clinical findings were misinterpreted and as a result the diagnosis of a pericardial effusion was missed.

HAEMATOLOGICAL FINDINGS

The haemoglobin levels and distribution have been recorded in table 15. The haemoglobin on admission ranged from 7.3 g. per cent (case 51) to 12.4 g. per cent (case 58) with an average of 10 g. per cent. Thirteen patients had a haemoglobin of less than 12 g. per cent and in 3 of these the Hb was less than 9 g. per cent. Peripheral smears in these patients showed a normocytic normochromic pattern.

On admission, 3 patients (cases 54,56 and 59) had a white cell count of 10,000 or less per c.mm., in 6 the count was between 10,000 and 20,000 per c.mm. and in 6 it was greater than 20,000 per c.mm.

The sedimentation rate (Wintrobe) was raised in every instance and ranged from 43 (case 62) to 64 mm. in one hour (case 65).

RADIOLOGICAL FINDINGS (See plates 9 - 13)

These have been recorded in table 16. Chest radiographs obtained on admission showed 12 patients to have enlargement of the transverse diameter of the heart, and in 9 of these the cardiac outline was globular in shape. Six patients (cases 51,55,59,60,61 and 64) showed massive cardiomegaly with a globular outline and a cardiothoracic

/ratio ...

TABLE 15

HAEMATOLOGICAL FINDINGS

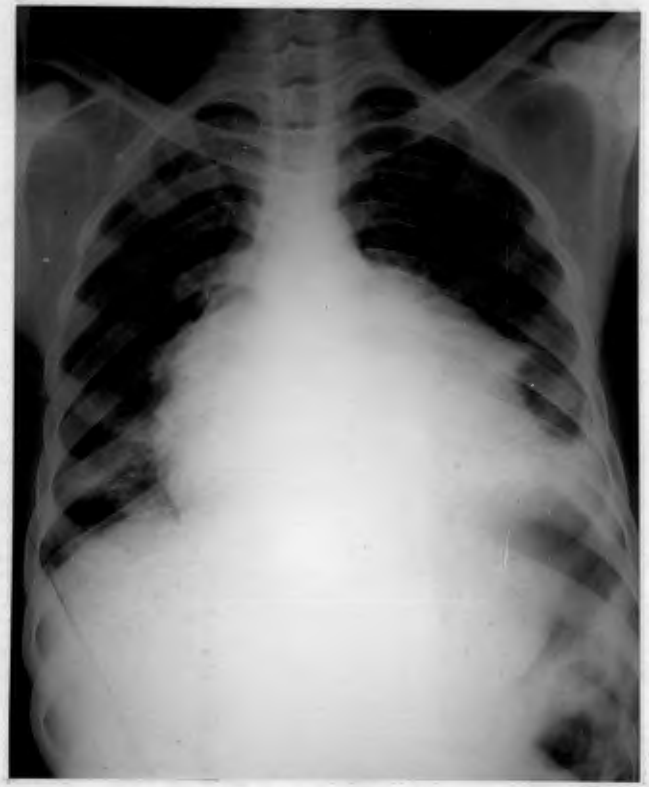
Hb g. per cent	No. of Patients	Per Cent
12 - 15	2	13
9 - 11.9	10	67
< 9	3	20

W.B.C. per c.mm.	No. of Patients	Per Cent
0 - 10,000	3	20
10,100 - 20,000	6	40
> 20,000	6	40

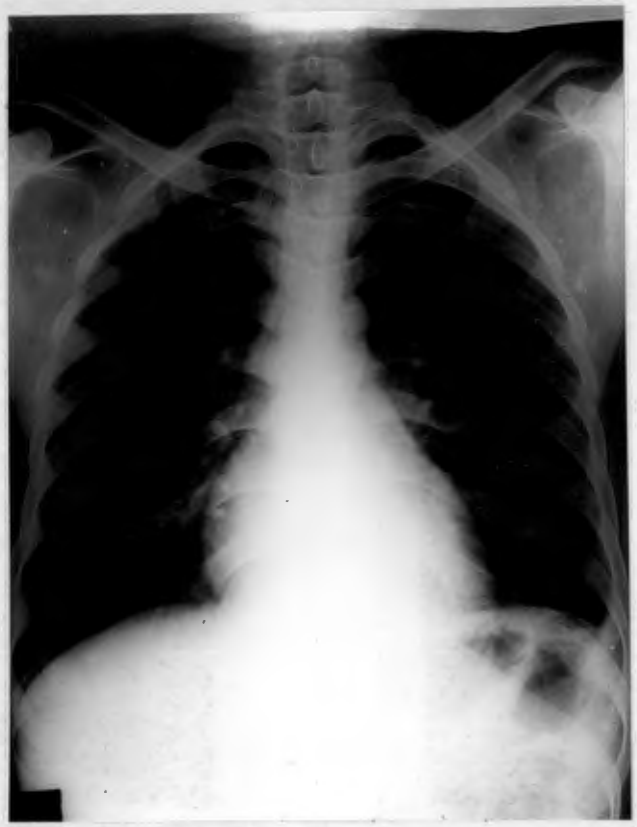
TABLE 16

RADIOLOGICAL FINDINGS IN 15 PATIENTS WITH AMOEBIC PERICARDITIS

Chest Radiograph	No. of Patients	Per Cent
Cardiothoracic ratio above 0.5	12	80
Cardiothoracic 0.7 or greater	6	40
Globular cardiac configuration	9	60
Localised bulging of left cardiac border	2	13
Elevated left diaphragm	4	27
Elevated right diaphragm	2	13
Pleural effusion left base	3	20
Pleural effusion right base	2	13
Chest Screening (14 patients)		
Decreased or absent cardiac pulsation	10	71
Immobility or decreased movement left diaphragm	7	50
Immobility or decreased movement right diaphragm	3	21

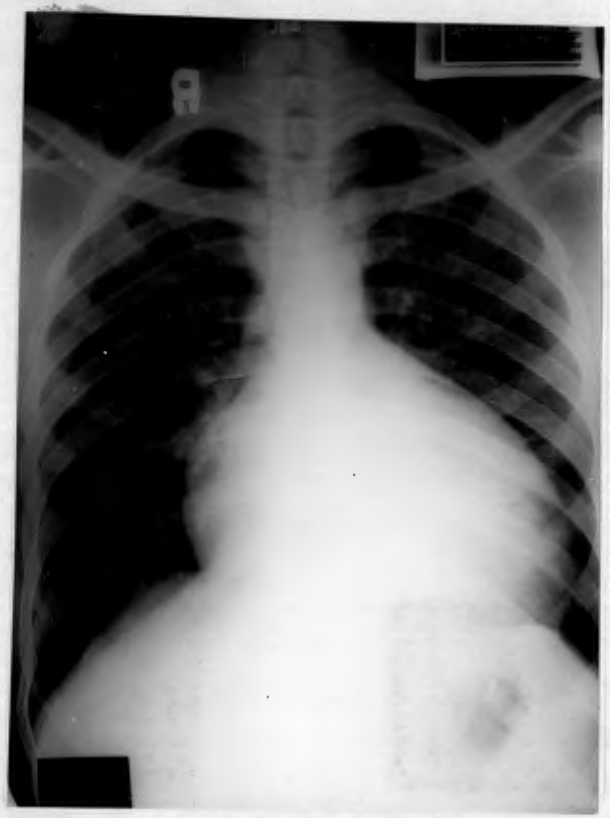


(a) Postero-anterior radiograph of chest showing globular cardiac contour produced by a large pericardial effusion.

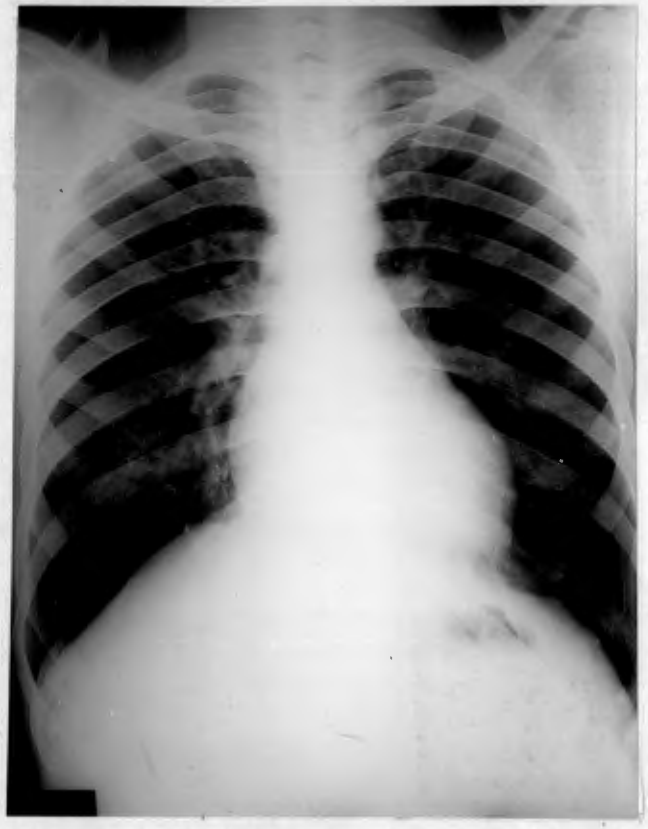


(b) Postero-anterior radiograph of the chest after 3 weeks of treatment showing normal cardiac size and contour

Plate 9 Case 55 A presuppurative amoebic pericardial effusion

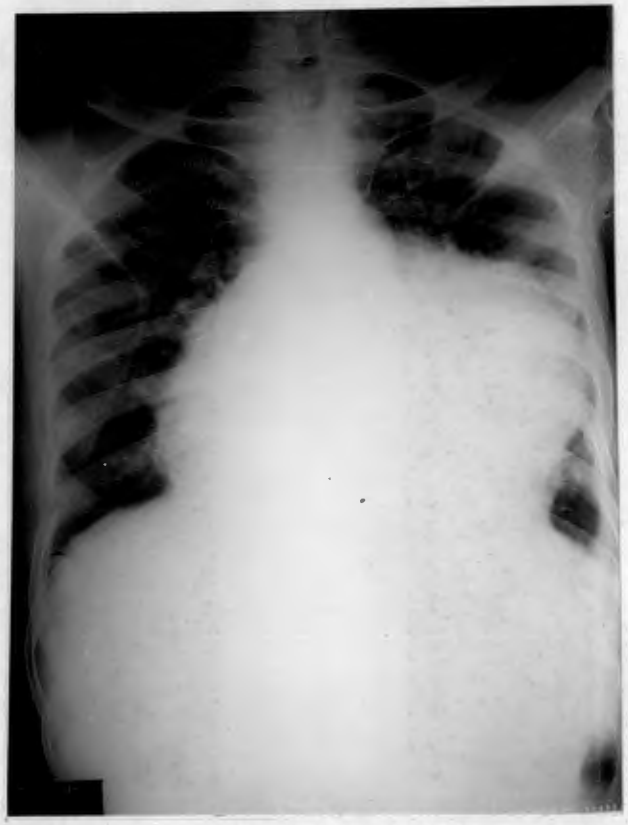


(a) Postero-anterior radiograph of chest showing changes in heart size and contour produced by a pericardial effusion

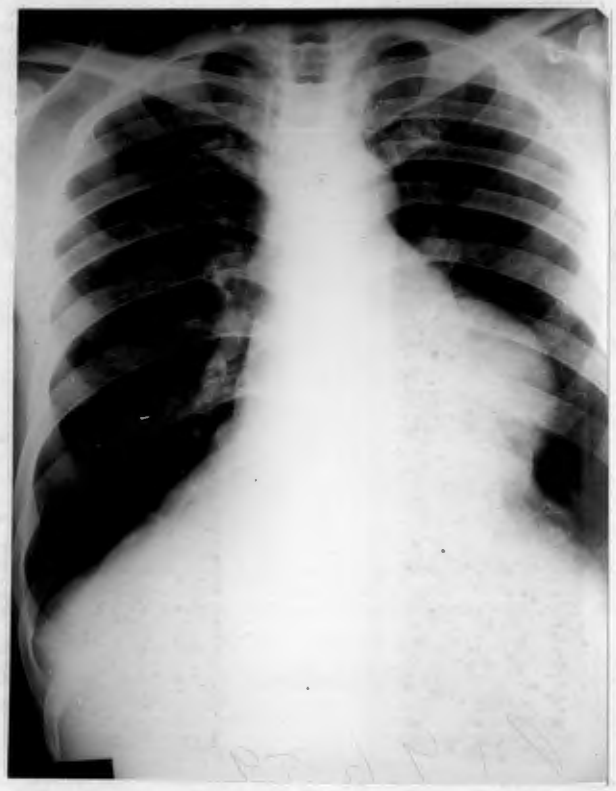


(b) Postero-anterior radiograph of chest after 4 weeks of treatment showing normal cardiac size and contour

Plate 10 Case 56 A suppurative amoebic pericardial effusion

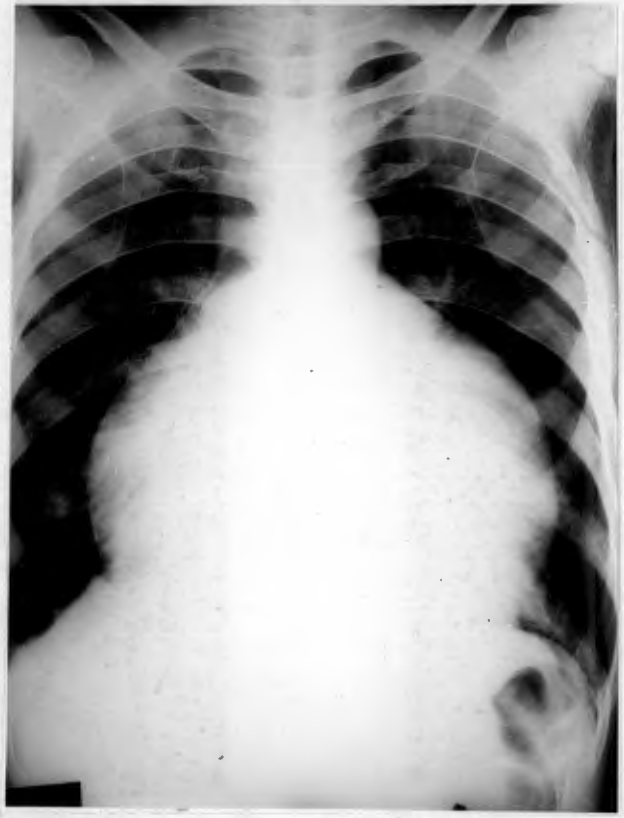


(a) Case 57 Massive increase in the heart shadow with a prominent bulge of the left cardiac border

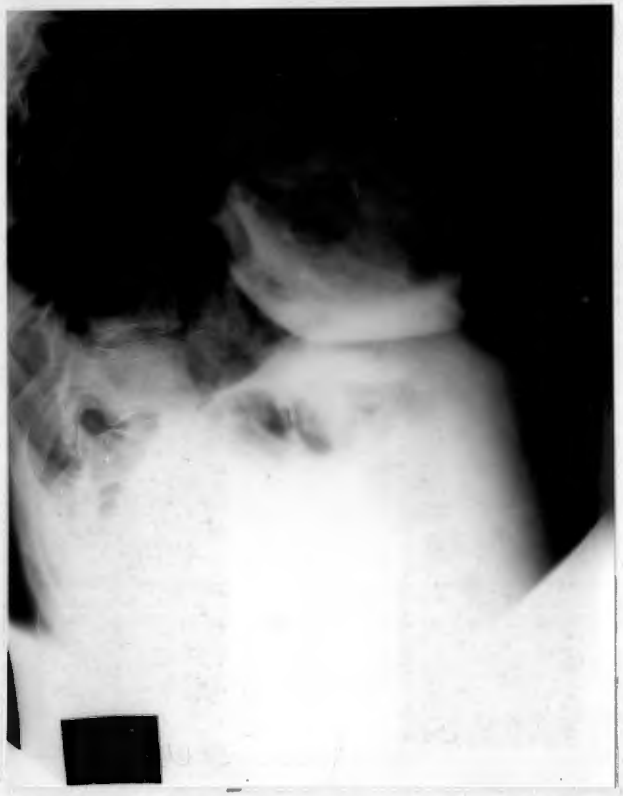


(b) Case 58 Localised bulge of the left cardiac border resembling a cardiac aneurysm

Plate 11 Postero-anterior radiographs of the chest showing deformities of the left cardiac border seen in 2 patients with suppurative amoebic pericardial effusions

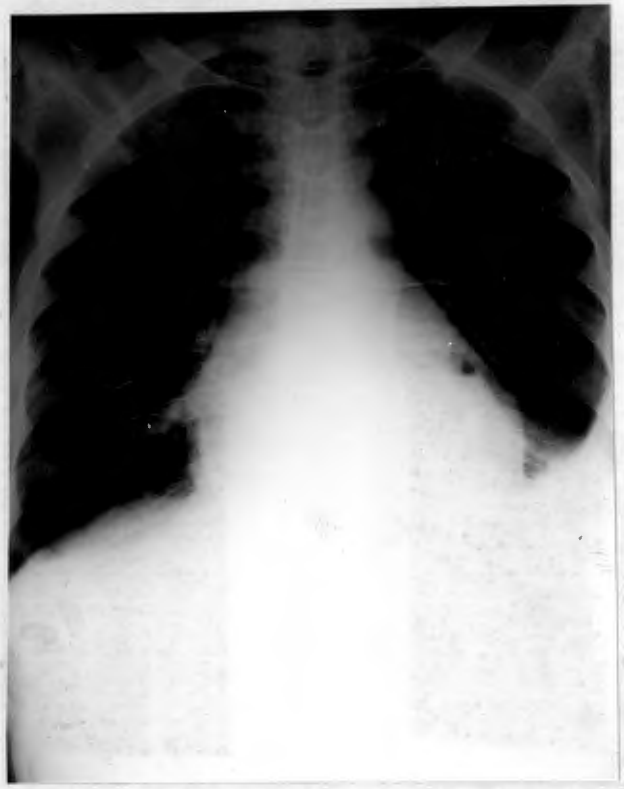


(a) Postero-anterior radiograph of the chest showing the changes in the heart size and contour produced by a large pericardial effusion

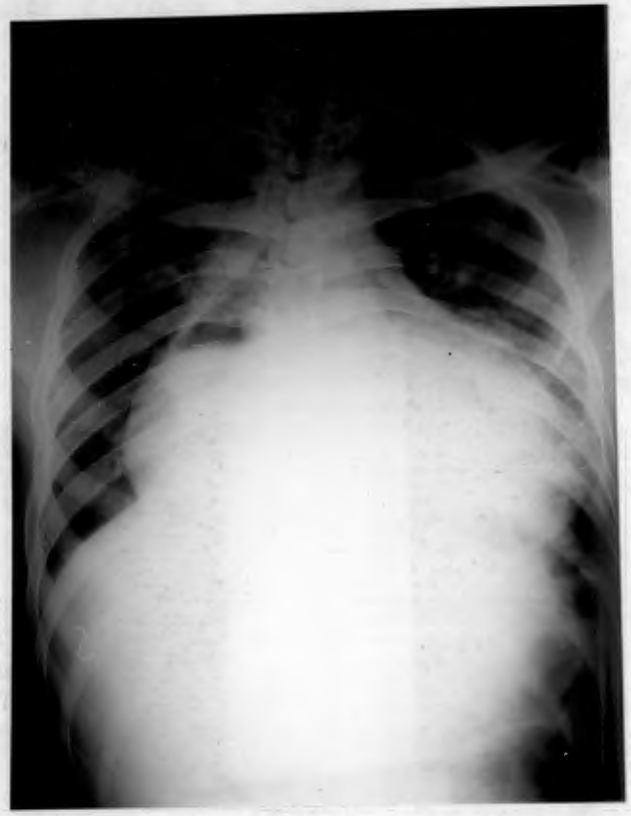


(b) Lateral radiograph of the chest showing the base of the heart outlined by lipiodol injected into the pericardial cavity and a subphrenic focus of lipiodol which has drained from the pericardial cavity.

Plate 12 Case 59 Radiographs demonstrating a hepatopericardial fistula in a patient with suppurative amoebic pericarditis



(a) Case 59



(b) Case 64

Plate 13 Postero-antero chest radiographs showing air-fluid levels in the pericardial cavity in suppurative amoebic pericarditis

ratio of 0.7 or more. Localised bulging of the left cardiac border was seen in 2 patients (cases 57 and 58).

Elevation of the left diaphragm was a feature in 4 patients (cases 54,55,56 and 64) and 2 (cases 56 and 62) showed elevation of the right diaphragm.

Small pleural effusions were seen at the left base in 3 patients (cases 55,56 and 65) and at the right base in 2 (cases 60 and 62). Shadows at the left base suggestive of atelectasis were noted in 2 instances (cases 54 and 64).

Chest Screening: Diminished or absent cardiac pulsation was present in 10 of the 14 patients screened. Immobility or diminished movement of the left diaphragm was seen in 7 and of the right diaphragm in 3.

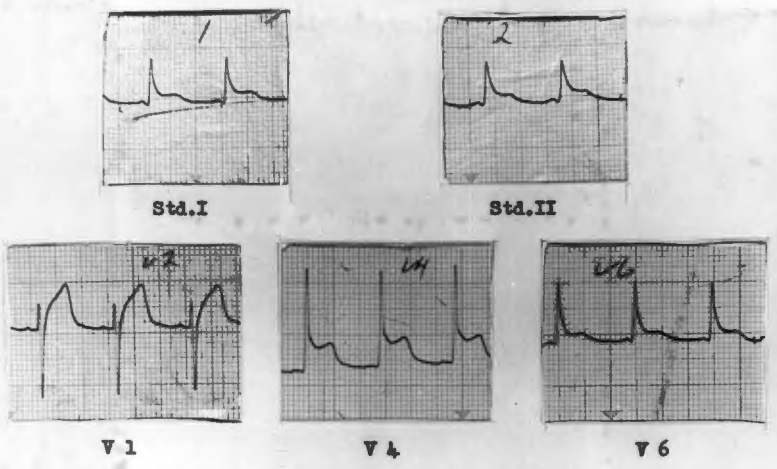
ELECTROCARDIOGRAPHIC FINDINGS (see plate 14)

The commonest electrocardiographic abnormality was generalised T-wave inversion seen in 11 patients. S-T segment elevation in either the standard leads, the augmented unipolar limb leads or the chest leads was present in 5 patients (cases 51,53,55,56 and 64), and low voltage patterns were seen in 2 (cases 59 and 60).

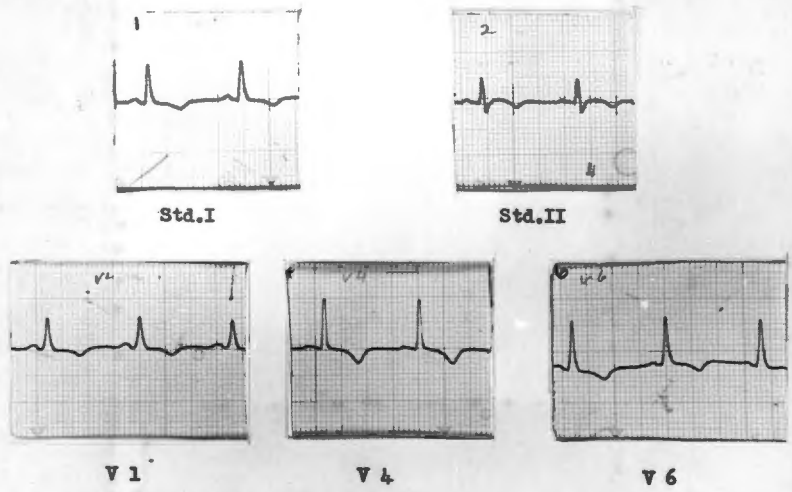
LIVER ASPIRATION

Aspiration of the left lobe of the liver via the epigastric route was attempted in 10 patients with success in 9 (cases 52,53,54,55, 56,60,63,64 and 65). A total of 25 diagnostic and therapeutic

/aspirations ...



CASE 53 ELECTROCARDIOGRAPH ON ADMISSION SHOWING GENERALISED S-T SEGMENT ELEVATION



CASE 53 ELECTROCARDIOGRAPH AFTER 2 WEEKS SHOWING GENERALISED T-WAVE INVERSION

Plate 14 Electrocardiographic changes in amoebic pericarditis

aspirations were performed yielding a total of 8,715 ml. of pus. Typical anchovy sauce pus was obtained from 7 patients (cases 52, 53,54,56,60,63 and 64), yellow pus was aspirated from one patient (case 55) and from the remaining patient (case 65) anchovy sauce pus and yellow pus were aspirated from different sites. A laparotomy localised a left lobe abscess in one instance (case 51) in which aspiration was unsuccessful.

Trophozoites of E.histolytica were identified in the aspirate of 2 patients (cases 54 and 60), and in the pus of one patient (case 51) with an external fistula. Staphylococcus pyogenes was cultured from the pus of one patient (case 60) and gram-negative bacilli found in the pus of another (case 55).

PERICARDIAL ASPIRATION

Pericardial aspiration was attempted in 11 patients with success in 10 (See table 17). Thirty diagnostic and therapeutic aspirations were performed yielding a total of 11,180 ml. of fluid. Typical anchovy sauce pus was aspirated from 6 patients (cases 56,59, 61,62,63 and 64), yellow pus from one patient (case 60) and sero-sanguineous fluid from 2 patients (cases 51 and 55). From the remaining patient serous fluid and subsequently blood-stained pus was aspirated (case 65).

The largest quantity of anchovy sauce pus obtained at a single aspiration was 1,500 ml. (case 61), and from one pericardial effusion

/(case 59)...

TABLE 17

CHARACTERISTICS OF THE PERICARDIAL ASPIRATE OF 10 PATIENTS

	No. of Patients	Per Cent
Anchovy sauce pus	6	60
Yellow pus	1	10
Serosanguineous fluid	2	20
Serous fluid (subsequently blood-stained pus)	1	10
E. histolytica identified in pus	3	30
Liver cells identified in pus	1	10
Sterile on culture for bacteria	9	90
Bacteria cultured	1	10

(case 59) which was aspirated 10 times a total of 6 litres of pus was removed.

Trophozoites of E.histolytica were identified in the pericardial pus of 3 patients (cases 59,61 and 64). Escherichia coli was cultured from the pus of one patient (case 59) and liver cells were identified in the pericardial aspirate of the same patient (case 59). Acid-fast bacilli were not found on direct examination or culture of the pericardial aspirates.

STOOL EXAMINATION

Trophozoites of E.histolytica were identified in the stools of one patient (case 56) and blood, pus and mucus in the stools of another (case 51). Two patients (cases 57 and 58) in whose stools E.histolytica were not found, were shown at necropsy to have amoebic ulceration of the colon.

DIAGNOSIS

Presuppurative Amoebic Pericarditis (See table 18): Five patients (cases 51 - 55) were considered to be suffering from non-suppurative or presuppurative amoebic pericarditis. In 2 (cases 51 and 55) the existence of a presuppurative pericarditis was proved by the aspiration of blood-stained serous fluid from the pericardium. In the remaining 3 (cases 52,53 and 55) the diagnosis of presuppurative rather than suppurative pericarditis was suggested by such findings as the less

/severe ...

TABLE 18

THE MAIN DIAGNOSTIC FINDINGS IN 15 PATIENTS WITH AMOEBIC PERICARDITIS

A. Presuppurative Group (cases 51 - 55)

(i) Findings suggesting presuppurative pericarditis

	Patients
Blood-stained serous fluid aspirated from the pericardium	2
The clinical presentation and response to treatment without drainage	3
TOTAL	5

(ii) Findings suggesting an amoebic liver abscess

	Patients
E.histolytica identified in the liver pus	2
Sterile anchovy sauce pus aspirated from the liver	2

severe clinical presentation and the response to specific treatment in the absence of pericardial drainage.

In 2 patients (cases 51 and 54) with presuppurative amoebic pericarditis the diagnosis of a left lobe amoebic liver abscess was proved by the identification of E.histolytica in the liver pus and in a further 2 (cases 52 and 53) it was strongly suggested by the aspiration of typical sterile anchovy sauce pus. In the remaining patient (case 55) an amoebic origin of the liver abscess was suggested by the aspiration of pus which was sterile on culture for bacteria.

Suppurative Pericarditis (See table 18): Ten patients (cases 56 - 65) were considered to be suffering from suppurative amoebic pericarditis. In 2 (cases 57 and 58) the diagnosis was established at necropsy. In 3 (cases 59,61 and 64) the diagnosis was proved by the identification of E.histolytica in pus aspirated from the pericardium and in a further 3 (cases 56,62 and 63) it was strongly suggested by the aspiration of typical sterile anchovy sauce pus. In the 2 remaining patients (cases 60 and 65) in this group the pericardial pus was not of a typical anchovy sauce character but was sterile on culture and aspiration of pus from an associated left lobe liver abscess suggested its origin.

The aspiration of pus from a left lobe liver abscess established the likely source of the suppurative amoebic pericardial effusions in 5 patients (cases 56,60,63,64 and 65). In two patients (cases 57 and 58) the left lobe liver abscess was only diagnosed at necropsy. The aetiology

/of ...

of the liver abscess was proved by the identification of E.histolytica in the liver pus of one patient (case 60) and was strongly suggested by the aspiration of typical sterile anchovy sauce pus from a further 4 patients (cases 56,63,64 and 65).

The diagnosis of the onset of constrictive pericarditis in 3 patients (cases 59, 60 and 62) was based on clinical, radiological and electrocardiographic findings. Signs of persisting tamponade and a well heard third heart sound when associated with serial radiographs showing a decreasing or normal heart size and chest screening showing decreased or absent pericardial pulsation formed the basis for the diagnosis. Two patients (cases 59 and 60) developed low voltage patterns in their electrocardiographic tracings.

TREATMENT

Fourteen patients in this group received the tissue amoebicides emetine hydrochloride and chloroquine (phosphate or sulphate) and 13 the intestinal amoebicide diiodohydroxyquinoline in addition. The dosage schedule was similar to that used in the pleuropulmonary group.

Treatment with emetine and chloroquine was begun on the 1st day in 13 patients. Unfortunately in one of these (case 58) treatment was discontinued on the 3rd day. One patient received emetine and chloroquine from the 6th day and one patient (case 58) was given chloroquine with no emetine on the 4th day.

One patient (case 53) proved to be of particular interest

/in ...

in that he was admitted on 3 occasions with a relapsing left lobe amoebic liver abscess and was given 4 full courses of emetine and chloroquine. A further patient (case 51) received a 2nd course of emetine and chloroquine following the relapse of a left lobe amoebic liver abscess which had been drained surgically.

In addition to specific therapy antibiotics were given to 12 patients. Prior to establishment of the diagnosis anti-tuberculous drugs in the form of streptomycin and isoniazid were mistakenly commenced in 2 patients (cases 53 and 52).

Seven patients (cases 53,57,58,60,61,64 and 65) were digitalised and received bi-weekly intramuscular injections of mersalyl.

CLINICAL PROGRESS

The progress of the 15 patients with amoebic pericarditis has been recorded in table 19. There were 4 deaths (cases 57,58,59 and 62) and 11 recoveries. Six patients who recovered (cases 56,60,61,63,64 and 65) had been shown by pericardial aspiration to be suffering from suppurative amoebic pericarditis and 2 (cases 51 and 55) from pre-suppurative amoebic pericarditis. The remaining 3 recoveries (cases 52,53 and 54) were regarded on clinical grounds as presentations of presuppurative amoebic pericarditis.

Three patients (cases 59,60 and 62) with suppurative amoebic pericarditis developed clinical features suggestive of constrictive pericarditis. In the first of these (case 59) a pericardectomy was

/performed ...

TABLE 19

CLINICAL PROGRESS OF 15 PATIENTS WITH AMOEBIC PERICARDITIS

Observations		Total No. of Patients Observed	Per Cent
Total deaths	4	15	27
Deaths from suppurative amoebic pericarditis	2	10	20
Deaths from constrictive amoebic pericarditis	2	3	67
Recovery from suppurative amoebic pericarditis	6	10	60
Recovery from presuppurative amoebic pericarditis	5	5	100
Average duration of pyrexia days after treatment	15	11	-
Average weight increase in pounds	13	8	
Duration of signs of pericarditis in days after specific treatment	19	9	
Follow-up 1 - 15 months			
Presuppurative amoebic pericarditis progress maintained	2	2	
Suppurative amoebic pericarditis progress maintained	6	6	

performed on the 69th day, in the second (case 60) on the 73rd day and in the remaining patient (case 62) on the 44th day. In each instance the diagnosis of constrictive pericarditis was verified at thoracotomy. Following attempted pericardectomy 2 (cases 59 and 62) of these 3 patients died. A further patient (case 65) developed features suggestive of a constrictive pericarditis but with continued observation these features gradually resolved.

The duration of hospitalisation in the 5 patients (cases 51, 52,53,54 and 55) with presuppurative amoebic pericarditis who recovered averaged 59 days, whilst in the 6 recoveries (cases 60,61,63,64 and 65) with suppurative amoebic pericarditis it averaged 88 days.

The average duration of the pyrexia following specific treatment in the 11 recoveries was 15 days. In spite of the fact that some patients were oedematous on admission there was an average weight increase of 13 lbs. in 8 of the 11 survivors in whom this was recorded.

Following specific treatment signs of pericardial involvement lasted for an average of 19 days in 9 of the 11 survivors. Of the remaining 2, one (case 60) developed constrictive pericarditis and signs of pericardial involvement cleared on the 90th day, 27 days after pericardiectomy, and in the other (case 65) slight elevation of the jugular venous pressure persisted at discharge and at follow-up clinic after one month.

HAEMATOLOGICAL PROGRESS

There was an average rise of 3.7 g. per cent in the haemoglobin of the 11 survivors during hospitalisation. The white cell count at discharge was 10,000 per c.mm. or less in 9 of these 11. In 8 patients in which serial sedimentation rates were obtained a considerable decrease in the rate was noted in each instance.

ELECTROCARDIOGRAPHIC PROGRESS

At the time of discharge generalised T-wave inversion persisted in the E.C.G. tracings of 7 of the 11 survivors. In 5 of these patients the E.C.G. pattern reverted to normal during a subsequent period of observation which varied from one to 6 months.

RADIOLOGICAL PROGRESS

Radiographs at discharge showed the heart size to be within normal limits in each of the 11 recoveries. Residual elevation of the left hemidiaphragm persisted in one (case 53) patient and residual elevation of the right hemidiaphragm in 2 (cases 51 and 56). Two patients (cases 53 and 61) had small residual effusions at the left base.

PROGRESS FOLLOWING DISCHARGE FROM HOSPITAL

Eight patients were observed for varying periods following discharge. Two (cases 51 and 53) diagnosed as presuppurative amoebic pericarditis attended for 15 and 10 months respectively. The first of

/these ...

these (case 51) was readmitted 3 weeks following discharge and treated for a relapsed left lobe amoebic liver abscess which had ruptured through the laparotomy scar and formed an external fistula. He subsequently recovered completely. The second patient (case 53) presented with a relapse of a left lobe abscess and pericarditis on two occasions during the above period and finally recovered after four courses of specific treatment and surgical drainage.

The 6 patients (cases 56,60,61,63,64 and 65) diagnosed as suppurative amoebic pericarditis were urged to attend follow-up clinic to exclude the possible onset of constriction. One attended for 12 months (case 60), 3 for 6 months (cases 56,61 and 64), one for 3 months (case 63) and one for one month (case 65). Each of these patients remained well, returned to his occupation and maintained clinical, radiological and electrocardiographic progress.

NECROPSY FINDINGS (See plate 15)

Necropsy was obtained in 4 patients (cases 57,58,59 and 62). At necropsy the diagnosis of suppurative amoebic pericarditis following rupture of a left lobe amoebic liver abscess first became apparent in 2 (cases 57 and 58) patients. One of these patients (case 57) was managed as a cardiac failure of undetermined origin, a left lobe liver abscess was suspected and chloroquine commenced but the patient died on the 4th day. In the other (case 58) a diagnosis of a left lobe amoebic liver abscess was made on admission. On the 3rd day this diagnosis was discarded in favour of one of subacute bacterial endocarditis with cardiac

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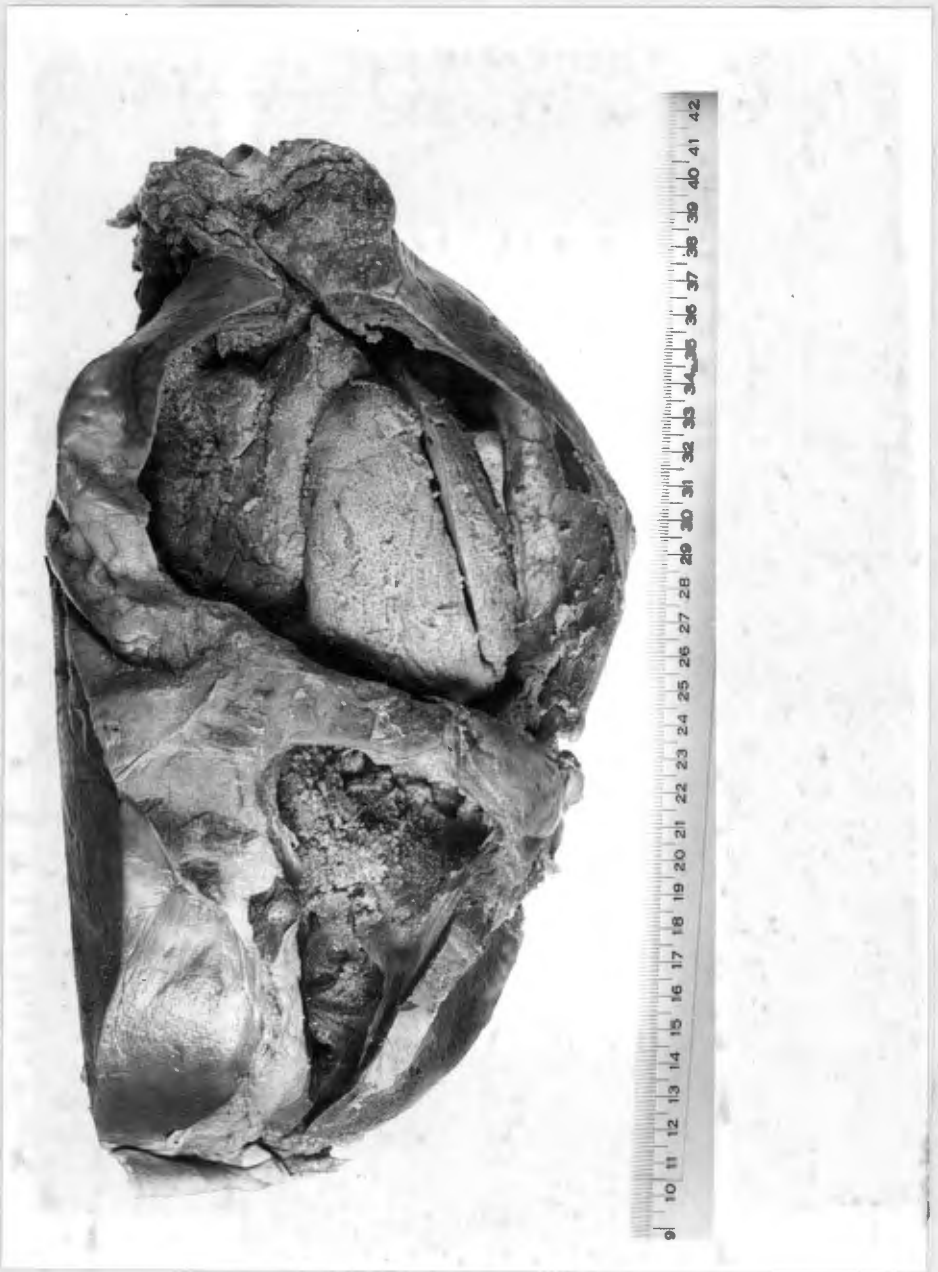

Plate 15 Case 58 Necropsy findings in suppurative amoebic pericarditis showing the heart and pericardial cavity and the responsible left lobe liver abscess. (scale in cms.)

failure and specific treatment was unfortunately discontinued.

Necropsy on the 2 patients diagnosed as constrictive amoebic pericarditis (cases 59 and 62) verified the diagnosis in each instance. In one (case 59) scar tissue in the left lobe of the liver represented the remains of a healed left lobe amoebic liver abscess which had ruptured into the pericardium. In the other (case 62) a large left lobe amoebic liver abscess with a hepatopericardial fistula was found with the constrictive pericarditis.

A BRIEF REVIEW OF THE LITERATURE WITH A DISCUSSION OF THE FINDINGS IN THIS STUDY

HISTORICAL REVIEW

"Behold, O Lord; for I am in distress:
my bowels are troubled; mine heart is turned
within me;

Lamentations,1, 20.

In 1860 Rouis working in Algeria published a study based on the necropsy findings in 162 patients with "tropical abscess" of the liver, in one of these, a left lobe liver abscess had ruptured into the pericardium. Other instances in which amoebic pericarditis was recognised at necropsy in the last century were reported by Zancarol (1893), Bertrand and Fontan (1895), and Howard and Hoover (1897).

It is claimed that Gremillon in 1889 was amongst the first to recognise a pericardial effusion associated with a liver abscess during life (Fiegel, 1959). His patient contracted dysentery in Indo-China and subsequently presented with the clinical features of a left lobe liver abscess and a pericardial effusion. Chocolate sauce pus was aspirated from the liver abscess and surgical drainage instituted. Necropsy revealed a suppurative pericardial effusion communicating through the diaphragm with a large left lobe liver abscess and "dysenteric" ulcers of the colon.

In 1908 Correlleur reported 3 patients shown to have suppurative amoebic pericarditis at necropsy. In one of these he recognised the condition during life and made an ante-mortem diagnosis of liver abscess of dysenteric origin complicated by a pericardial effusion.

Lasnse in 1916 appears to have been the first to identify amoebae in pericardial pus. He aspirated similar pus from a left lobe liver abscess and pericardial effusion and identified amoebae in both aspirates (Lasnse and Melnotte, 1928).

Piessinger and Casteran (1927) reported the first patient to recover from presuppurative amoebic pericarditis. Whitish-yellow fluid containing leucocytes was aspirated from the pericardium and chocolate pus from a left lobe liver abscess. Lipiodol was injected into the liver abscess but no communication demonstrated between this abscess and the pericardial effusion. Their patient recovered completely following repeated aspirations of the liver abscess and treatment with emetine.

In 1935 Huard and Meyer-May reported the first recovery from suppurative amoebic pericarditis. Sterile brown pus was aspirated from the pericardium of their patient who recovered following surgical drainage of the responsible liver abscess and treatment with emetine and stovarsol.

INCIDENCE

Reviewing the literature from 1885 to 1950 Carter and Koronas (1950) were able to find 44 reports of suppurative amoebic pericarditis and a further 14 reports in which the description was apparently that of presuppurative amoebic pericarditis. Six years later with a personal case report and a further 2 from the literature Norris and Beemer (1956) raised the total to 47 cases of suppurative amoebic pericarditis, a figure quoted by Takaro and Bond (1958) in their review. Table 20 shows the reported frequency with which amoebic pericarditis occurs as a complication of amoebic liver abscess. It is apparent from the figures quoted that this is a very rare condition and an uncommon complication of amoebic liver abscess.

AGE AND SEX

Fiegel (1959) discussing 34 case reports of amoebic pericarditis collected from the literature noted that the highest incidence occurred in the third and fourth decades. Laha (1946) and Stephan (1948) documented case reports of amoebic pericarditis of a non-suppurative variety in females. These appear to be the only two instances in which amoebic pericarditis has been reported in a female during the past 60 years, a finding which is in keeping with the lesser frequency of amoebic liver abscess in females (DeBakey and Ochsner, 1951).

In my patients amoebic pericarditis occurred most frequently in the third and fourth decades. During this study only one female

/with ...

TABLE 20

THE REPORTED FREQUENCY WITH WHICH AMOEBIC PERICARDITIS COMPLICATES AMOEBIC LIVER ABSCESS

Author	Liver Abscesses	Pericarditis	Per Cent
Rouis (1860)	162	1	.6
Justi (1923)	689	19	2.8
Petridis and Zancarol (1925)	731	3	.4
Huard and Meyer-Hay (1936)	150	3	.2
Ochsner and DeBakey (1943)	181	1	.6
Lamont and Peeler (1958)	250	7	2.8

with amoebic pericarditis, presumed to be of a presuppurative nature, was seen. Signs of pericarditis resolved following aspiration of the liver abscess and specific treatment. The details of her clinical presentation have not been included.

PATHOGENESIS

PRESUPPURATIVE AMOEBIC PERICARDITIS

Verges and Hermenjat-Gerin (1932) stated that like the pleural membranes the pericardial membranes could react to neighbouring amoebic pathology in the liver with the formation of a serous effusion.

Analysing 43 necropsies of liver abscesses of variable etiology Huard and Meyer-May (1936) found serous pericardial effusions in six. Included in this group was a patient with amoebic colitis, a left lobe amoebic liver abscess which had ruptured into the right lung and a large serous pericardial effusion. Discussing the migration of left lobe liver abscesses into the thorax these authors described the following stages. Firstly, the appearance of pericarditis or a serous pericardial effusion resulting from the adjacent inflammation or "reaction de voisinage". The clinical details of a patient with this variety of amoebic pericarditis were documented. Secondly, with more intensive involvement of the pericardial layers an aseptic purulent effusion "pericardite puriforme aseptique" may develop in the absence of frank rupture. These workers described such a patient in whom they were unable to demonstrate a hepatopericardial fistula at surgery. Finally, frank rupture of the liver abscess into the pericardial cavity may occur with resultant suppurative amoebic pericarditis.

Edwards (1947) described a patient suffering from presuppurative

/amoebic ...

amoebic pericarditis, which he considered to have resulted from adjacent pulmonary amoebiasis in the right lung.

Coirault et al. (1955) reported a patient with non-suppurative amoebic pericarditis resulting from an adjacent amoebic abscess of the left lobe of the liver. Describing the effusion these workers used the terms "sympathetic" and "reactive". The term sympathetic was also used by Gordon (1956) and Lamont and Pooler (1958) to describe the non-suppurative or presuppurative presentation of amoebic pericarditis. Lamont and Pooler (1958) also noted that the initial sympathetic or slightly purulent effusion may be the first phase in the evolution of amoebic pericarditis being followed by intra-pericardial rupture of the responsible amoebic liver abscess.

D'Mello (1947) described histological findings which suggested that in his patient there had been invasion of the epicardium by E.histolytica some days prior to frank rupture of the liver abscess.

There were 5 patients in the present study considered to be suffering from presuppurative amoebic pericarditis resulting from pericardial involvement by the inflammatory process at the periphery of an extending left lobe amoebic liver abscess. In 2 of these (cases 51 and 55) serosanguineous fluid was aspirated from the pericardium whilst in the remaining 3 the diagnosis was made on clinical grounds. A further patient who initially presented with a serous pericardial effusion subsequently progressed to develop a suppurative amoebic pericarditis (case 65).

The entity of presuppurative amoebic pericarditis nearly always results from involvement of the pericardial layers in the reaction at the periphery of an extending amoebic liver abscess. The nature of the pericardial fluid may be serous or seroSanguineous. The term "sympathetic" when used to describe these effusions is unsatisfactory (Wilmot, 1962). For lack of a better descriptive terminology the designation presuppurative or non-suppurative is preferred to distinguish these effusions from suppurative amoebic pericardial effusions following frank rupture of an amoebic liver abscess. As mentioned previously it is possible that when the adjacent inflammatory reaction is severe that an amoebic pericardial effusion can be of a purulent nature in the absence of frank rupture of the responsible liver abscess.

SUPPURATIVE AMOEBIC PERICARDITIS

In 1932 Verges and Hermenjat-Gerin collected 80 case reports from the literature describing amoebic liver abscesses which had ruptured into serous cavities. Included in their series were 13 of suppurative amoebic pericarditis. In each of these an amoebic liver abscess of the left lobe of the liver formed the source of the pericardial lesion and spread had occurred by direct extension through the diaphragm into the pericardial sac.

Huard and Meyer-May (1936) in an impressive treatise entitled "Les Abcès du Foie" discussed the migratory pathways of a left lobe liver

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abscess. They quoted case reports illustrating extension of a left lobe liver abscess into the sub-diaphragmatic space, peritoneal cavity, stomach and other abdominal viscera. They noted that transdiaphragmatic extension may take place into the pericardium, pleura, lungs or mediastinum. Of particular interest were their illustrations showing that in the same patient extension may occur into the pericardium and associated structures. They quoted Graves (1839) as describing concomitant migration into the pericardium and stomach, Corrolleur (1908) who recorded migration into the pericardium and right lung, Sanbus (1911) who recorded migration into the pericardium and inferior vena cava, Scuderi (1928) who recorded migration into the pericardium and right pleura and Chatterji (1927) who described migration into the pericardium and ventricle of the heart.

Recent reports also indicate that suppurative amoebic pericarditis almost always results from extension or rupture of a left lobe amoebic liver abscess (Carter and Korones, 1950; Morris and Boomer, 1956; Lamont and Pooler, 1958).

Rarely amoebic pericarditis may result from involvement of the pericardium by an abscess situated in the right lobe of the liver (Huard and Meyer-May, 1936; Kern, 1945; Laha, 1946). Huard et al. (1933) and Schrire (1959) each encountered a patient in whom suppurative pericarditis may have resulted from contiguous spread of an adjacent lung abscess. In each of these it was impossible to ascertain to what extent

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the amoeba was implicated in the pathogenesis. In the patient reported by Huard et al. (1933) the lung abscess and pericarditis contained pyogenic organisms whose probable origin was a secondarily infected amoebic liver abscess. Buri et al. (1955) reported a patient who presented with a left-sided amoebic empyema and subsequently developed a pneumopyopericardium from which pus and large quantities of air were aspirated. Necropsy revealed a left-sided empyema with a pleuropericardial fistula and left lobe amoebic liver abscess with a hepatopericardial fistula.

The high mortality associated with suppurative amoebic pericarditis has provided ample opportunity to study the pathology of direct extension of an amoebic liver abscess into the pericardium. At necropsy the superior surface of the left lobe of the liver, the diaphragm and the parietal pericardium are bound together by a mass of adhesions (Verges and Hermenjat-Gerin, 1932; Huard and Meyer-May, 1936; Lamont and Peeler, 1958). Fistula connecting the liver abscess and pericardial cavity may be single or multiple and vary in size from one to several centimeters in diameter (Verges and Hermenjat-Gerin, 1932; Huard and Meyer-May, 1936).

Clinical and pathological findings in this study suggested that all cases of suppurative amoebic pericarditis resulted from extension of a left lobe liver abscess into the pericardium. In one patient following intra-pericardial rupture further extension took place into the mediastinum and left pleural cavity (case 58). Necropsy findings in 4 patients (cases 57,58,59 and 62) showed extensive adhesions between

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the superior surface of the liver and diaphragm with a hepatopericardial fistula in each instance.

CONSTRICTIVE AMOEBIC PERICARDITIS

Parison et al. (1958) noted that in their patient with amoebic pericarditis neither adhesive constrictive pericarditis nor other sequelae followed surgical drainage. Lamont and Peeler (1958) noted that 3 of their patients with suppurative amoebic pericarditis, following surgical drainage of the pericardium, subsequently progressed to a stage of constriction and died. Evidence of constriction was found at necropsy in each instance. Schrire (1959) quoted by Wilmot (1962) described a patient, in whom the diagnosis of suppurative amoebic pericarditis was presumptive, who developed, "acute severe pericardial constriction," which required surgery. Following observation of 2 of the patients reported in this study Wilmot (1962) concluded that although constrictive pericarditis may follow amoebic pericarditis, it was as yet uncertain whether secondary bacterial infection of the pus was a necessary factor.

There were 3 patients (cases 59,60 and 62) in this study who developed constrictive pericarditis. In 2 of these as suggested by Wilmot (1962) secondary bacterial invasion of the pericardium may have been a factor in determining the onset of constriction. In the remaining patient (case 62) the pericardial pus was sterile on culture and there was no evidence of bacterial pericarditis at necropsy. I have since seen a further patient with suppurative amoebic pericarditis, in whom there was

/no ...

no evidence of bacterial invasion of the pericardium, progress to a stage of constriction.

CONCLUSIONS

Amoebic pericarditis is a rare complication of an amoebic liver abscess. To date only 2 females with this complication have been reported in the literature. Prior to frank rupture, the inflammatory reaction surrounding an amoebic liver abscess may involve the pericardium and produce a pericarditis with or without a serous or serosanguineous effusion. Rarely, in the absence of frank rupture or a demonstrable hepatopericardial fistula suppurative amoebic pericarditis may result from the above reaction.

Suppurative amoebic pericarditis almost always results from rupture or extension of an abscess of the left lobe of the liver. Rarely extension may occur from an abscess in the right lobe of the liver or from an adjacent amoebic focus in the lung or pleura.

Patients who survive the rupture of an amoebic liver abscess into the pericardium may subsequently develop constrictive pericarditis. Though secondary bacterial invasion of the pericardium may be a factor in determining the onset of constriction, patients have been seen to progress to a stage of constriction in whom there was no evidence of this.

THE CLINICAL FINDINGS IN AMOEBIC PERICARDITIS

CONSTITUTIONAL SYMPTOMS AND SIGNS

Reports in the literature indicate that fever, profuse sweats, weakness and weight loss are common constitutional symptoms. On examination, a low grade pyrexia, evidence of weight loss, cachexia or clinical anaemia are commonly found (Carter and Kerenes, 1950; Norris and Beemer, 1956; Lamont and Pooler, 1958).

The constitutional symptoms and signs associated with amoebic pericarditis in the present study were similar to those mentioned above. It can be seen that they were also similar to those found in the pleuro-pulmonary group and that clinical anaemia was a frequent finding.

AMOEBIC LIVER ABSCESS OF THE LEFT LOBE

In many reports of amoebic pericarditis the initial symptoms and signs have been those of a left lobe liver abscess (Gerdes, 1956; Norris and Beemer, 1956; Lamont and Pooler, 1958). In a recent study of 8 patients with left lobe amoebic liver abscess Alkan et al. (1961) recorded the main clinical features. Pain situated in the epigastrium or left hypochondrium, sometimes referred to the left shoulder, was the major symptom and the usual local findings were enlargement of the left lobe of the liver with epigastric tenderness. They found physical signs at the left lung base in 3 of their patients. A superficially situated left sided amoebic liver abscess may present as a visible tender epigastric swelling as was the case in 3 of the 4 patients described by

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Ghosh (1954). This worker also stressed that abscesses of the left and right lobes may occur concurrently in the same patient. Other workers who have described the clinical syndrome of an amoebic abscess of the left lobe of the liver have been Bressot (1930), Lamont and Pooler (1958) and Paul (1960).

Whilst an amoebic liver abscess of the left lobe of the liver is the common precursor of amoebic pericarditis there have been occasional case reports in which an amoebic abscess of the right lobe of the liver was responsible (Kerm, 1945; Laha, 1946; Stephan, 1948; Lamont and Pooler, 1958). In such presentations the initial symptoms and signs are those of an amoebic abscess of the right lobe of the liver with the subsequent onset of manifestations of pericarditis.

The majority of my patients presented with clinical findings compatible with an amoebic liver abscess of the left lobe in addition to those of a pericardial effusion. A liver abscess and a pericardial effusion may each produce a tender hepatomegaly, and because of this in a patient with a pericardial effusion the liver abscess may be overlooked. In my patients features such as left shoulder tip pain, an area of localised epigastric tenderness and guarding or swelling suggested the presence of an abscess in addition to congestion of the liver.

There were 2 patients (cases 57 and 58) in this study in whom the significance of the abdominal findings was not appreciated and as a result the presence of a left lobe liver abscess was missed.

Five patients (cases 51,52,56,63 and 65) had in addition to local findings compatible with a left lobe liver abscess, signs at the right base consistent with an associated abscess of the right lobe of the liver.

PRESUPPURATIVE AMOEBIC PERICARDITIS

Clinical reports of presuppurative amoebic periarditis have been documented by Laigret (1928), Huard and Meyer-May (1936), Purcell (1938), Staffieri et al. (1944), Laha (1946), Edwards (1947), Stephan (1948), Coirault et al. (1955) and Lament and Peeler (1958).

The above reports indicate that in addition to the symptoms of an amoebic liver abscess previously mentioned that extension of the epigastric pain to a retrosternal position with accompanying dyspnoea may herald the onset of amoebic periarditis. Clinical findings resulting from adjacent amoebic pathology may vary in degree from a pericardial rub (Edwards, 1947) to signs of a pericardial effusion with minimal to moderate tamponade (Stephan, 1948; Lament and Peeler, 1958). Signs of severe tamponade were present in the patient reported by Coirault et al. (1955).

A noteworthy feature in the above case reports was that the clinical picture was frequently complicated by signs and symptoms suggesting involvement of viscera and serous cavities other than the pericardium. The patient reported by Edwards (1947) had signs and symptoms of a right-sided liver abscess with a hepatobronchial fistula.

Stephan (1948) found signs of a right pleural effusion with an underlying liver abscess and aspirated chocolate pus from the liver abscess and serous fluid from the pericardium. In the patient described by Helluy et al. (1949) the clinical presentation was that of a polyserositis, and the responsible left lobe liver abscess was drained at laparotomy. Coirault et al. (1955) were fortunate in that their patient suddenly developed a large left effusion and the chocolate pus aspirated suggested the diagnosis.

In some instances clinical evidence of the amoebic abscess responsible for pericardial involvement may be minimal or absent. Lamont and Pooler (1958) were forced to resort to exploratory puncture of the left lobe of the liver in one of their patients in order to demonstrate the responsible abscess. In the early stage of the illness in the patients described by Helluy et al. (1949) and Coirault et al. (1955) there was little to suggest that a left lobe abscess was responsible for the pericardial effusions.

The clinical features of presuppurative amoebic pericarditis in this study (cases 51 - 55) have been recorded. In each of these 5 patients symptoms and signs compatible with an abscess of the left lobe of the liver were associated with those of pericarditis. In 2 (cases 51 and 52) there were signs compatible with an additional abscess of the right lobe of the liver. Signs of pericarditis varied from a pericardial rub to those of a pericardial effusion with evidence of

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moderate to severe tamponade. With the exception of small associated pleural effusions the clinical presentation in my patients was not complicated by extension of the liver abscess to involve serous cavities or viscera other than the pericardium.

SUPPURATIVE AMOEBIC PERICARDITIS

Descriptions of the acute onset of amoebic pericarditis have been documented by Singh (1946), D'Mello (1947), Wilmot (1949), Harts (1950), Gordon (1956), Norris and Beemer (1956), Hollender and Grenier (1957), Lamont and Pooler (1958) and Singh and Jolly (1962). Rupture of an amoebic liver abscess into the pericardium may cause severe shock and death within hours but more commonly the onset is less abrupt (Wilmot, 1962). Extension of pain experienced in the epigastrium to the retrosternal region may occur with the onset of dyspnoea. On examination the most striking features are those of shock and an anxious distressed patient is seen with a subnormal temperature, cold clammy skin, feeble peripheral pulses and a low blood pressure. In the patients reported by D'Mello (1947), Wilmot (1949) and Harts (1950) death occurred in this acute phase of peripheral vascular collapse. In the patient reported by Parison et al. (1958) features suggesting pericardial irritation preceded the acute episode of rupture.

Rarely, prodromal clinical features suggestive of an amoebic liver abscess such as a history of epigastric pain, localised epigastric tenderness and a tender hepatomegaly are absent. In such instances

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following rupture of the clinically silent liver abscess the acute findings recorded above may be the presenting features as in the patient reported by Sambuc (1911).

Patients reported by Gordon (1956), Hollender and Grenier (1957), Lamont and Pooler (1958) and Singh and Jolly (1962) survived the initial episode of rupture or extension of the liver abscess into the pericardium. In each of these patients signs of a pericardial effusion with moderate to severe tamponade were detected. Features such as tachycardia, a thready peripheral pulse, pulsus paradoxus and raised neck veins were noted. On examination of the heart, enlargement of the area of cardiac dullness, an impalpable apex beat, distant heart sounds, a gallop rhythm and pericardial friction rub were the usual findings. In the patients reported by Gordon (1956) and Singh and Jolly (1962) and in some reported by Lamont and Pooler (1958) cardiac tamponade was severe and only relieved by immediate aspiration. Death may occur in this stage from progressive tamponade as in the patient reported by Norris and Beemer (1956).

Patients in whom the onset of suppurative amoebic pericarditis has been less abrupt have been reported by Kern (1945), Carter and Koronos (1950) and Alkan et al. (1961). In such presentations substernal pain and increasing dyspnoea may be superimposed on symptoms of a left lobe liver abscess. The clinical signs of a pericardial effusion have a more gradual onset. Rarely, with a clinically silent liver abscess the presentation may be that of a pericardial effusion

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of gradual onset. In the patients reported by Kern (1945) and Carter and Koronas (1950) clinical features compatible with a liver abscess preceded the onset of pericardial effusions but their significance was not appreciated.

Rarely, as in the patient reported by D'Mello (1947), signs and symptoms of acute dysentery may be associated with the presentation of amoebic pericarditis.

Fortunately, each of the 8 patients in this study in whom the diagnosis of suppurative pericarditis was established survived the episode of intrapericardial rupture or extension. In this respect their presentation was similar to that in the patients reported by Gordon (1956), Hollender and Grenier (1957), Lamont and Pooler (1958) and Singh and Jolly (1962) mentioned above. Whilst signs of severe cardiac tamponade were present in each of these 8 patients and were responsible in most for admission to hospital, it was often impossible to determine the degree of rapidity with which they developed. As judged from the histories obtained there were few in which the onset was dramatic. In one patient the presentation was similar to that reported by Gordon (1956) in that signs of a pericardial effusion developed whilst under observation in hospital and in this instance the period of onset was less than 24 hours.

Symptoms and signs referable to the epigastrium recognised or suspected as being compatible with a left lobe amoebic liver abscess were found in each of the 8 patients in this study in whom the diagnosis

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of suppurative amoebic pericarditis was established. In this respect my patients were similar to those reported by Gordon (1956), Hollender and Grenier (1957); Lament and Pooler (1958) and Singh and Jolly (1962). A significant finding was that 3 of these 8 patients had clinical signs at the right lung base compatible with a second abscess in the right lobe of the liver.

The diagnosis of suppurative pericarditis was missed in 2 of my patients. In the one (case 57) it was not appreciated that the signs of peripheral vascular collapse were due to a pericardial effusion until the patient was terminal. In the other (case 58) the cardiac findings were atypical and the possibility of a pericardial effusion was not considered. Both these patients were similar to those reported by Kern (1945) and Carter and Korones (1950) in that the presence of a liver abscess was not suspected.

CONCLUSIONS

Constitutional symptoms and signs in amoebic pericarditis are similar to those resulting from an amoebic liver abscess and include manifestations such as fever, sweating, weakness, a low grade pyrexia, evidence of weight loss and clinical anaemia.

The only distinctive findings in amoebic pericarditis may be clinical findings compatible with an abscess of the left lobe of the liver. These may be absent, or, when present, may be easily overlooked and mistaken for signs of hepatic congestion resulting from the pericardial effusion. In the clinical presentation of

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amoebic pericarditis associated with an abscess of the left lobe of the liver, symptoms and signs of a second abscess of the right lobe of the liver may be present. Rarely, in the absence of signs of a left lobe abscess, signs suggestive of abscess of the right lobe of the liver may be associated with those of pericarditis.

Presuppurative amoebic pericarditis may present with a pericardial friction rub or signs of a pericardial effusion with or without evidence of moderate or severe cardiac tamponade. Cardiac tamponade is usually not as severe in presuppurative as in suppurative amoebic pericarditis.

Suppurative amoebic pericarditis may present suddenly with severe retrosternal pain and signs of peripheral vascular collapse. Signs of shock may dominate the presentation. More often the onset is less dramatic and signs of a pericardial effusion with severe cardiac tamponade are found in a patient with a clinical history and findings compatible with a left lobe amoebic liver abscess. Occasionally signs compatible with presuppurative amoebic pericarditis such as a pericardial friction rub may precede the onset of suppurative amoebic pericarditis. Rarely there may be signs of concomitant intestinal amoebiasis.

INVESTIGATIONS IN PERICARDIAL AMOEBIASIS

HAEMATOLOGICAL FINDINGS

Haemoglobin: Haemoglobin levels consistent with a moderate to severe anaemia were present in patients reported by Carter and Korenes (1950), Buri et al. (1955), Gordon (1956) and Lamont and Pooler (1958). In Gordon's patient, following treatment with emetine and chloroquine, the haemoglobin rose from 5 g. to within normal limits.

Moderate to severe anaemia of a normochromic normocytic type was frequently present in my patients. A rise in the haemoglobin was seen in a number of patients following treatment with emetine and chloroquine but in others the response to these drugs could not be assessed as blood transfusions were given. It is probable that the anaemia in amoebic pericarditis results from the necrotic process in the liver and pericardium and that it is similar in this respect to the anaemia of infection associated with a chronic suppurative focus.

Leucocytosis: Leucocyte counts ranging from 8,600 per c.mm. (Edwards, 1947) to 39,000 per c.mm. (Kern,1945) have been reported but in the majority of patients with amoebic pericarditis a moderate leucocytosis is the rule (Carter and Korenes, 1950; Buri et al., 1955; Michon et al. 1959). In most instances the differential count showed an increase in the percentage of polymorphonuclear cells (Buri et al., 1955; Gordon, 1956; Michon et al., 1959) but in the patient reported by Carter and Korenes (1950) there was a relative increase in the lymphocytes.

Somewhat surprisingly 6 of the 15 patients in this study had a leucocyte count of greater than 20,000 per c.mm. whilst in 3 it was less than 10,000 per c.mm. Differential counts when obtained showed a variable increase in the percentage of polymorphonuclear cells. The leucocyte count in pericardial amoebiasis may be within normal limits, moderately elevated or markedly elevated and for this reason in my experience has seldom been of assistance in diagnosis.

Sedimentation Rate: Sedimentation rates of more than 60 mm. per hour were found in the patients reported by Kern (1945), Gordon (1956), Alkan et al. (1961). Recovery in Gordon's patient was associated with return of the sedimentation rate to within normal limits.

The sedimentation rate was invariably increased in my patients with amoebic pericarditis. Recovery was accompanied by a decrease in the rate in each instance.

RADIOLOGICAL FINDINGS

The experience of Edwards (1947) has shown that the early stages of amoebic pericarditis may be associated with no radiological changes in the heart size or contour. However, the majority of patients reported with presuppurative lesions have shown changes compatible with a moderate to large pericardial effusion (Purcell, 1938; Stephan, 1948; Cairault et al., 1955; Lamont and Pooler, 1958).

Reports in the literature indicate that suppurative amoebic

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pericarditis almost invariably produces radiological changes in the cardiac silhouette compatible with a pericardial effusion (Kern, 1945; Carter and Kerenes, 1950; Hollender and Grenier, 1957; Lamont and Peeler, 1958). Takaro and Bond (1958) noted that amoebic pericarditis is often characterised radiologically by a large cardiac shadow especially of a globular or "water bottle type".

The above remarks indicate that the radiological changes in the cardiac outline in amoebic pericarditis are non-specific. A noteworthy feature in some of the above reports, however, was that though chest radiographs initially showed a normal cardiac outline changes compatible with a pericardial effusion subsequently developed soon after admission in association with an acute clinical episode (Kern, 1945; Carter and Kerenes, 1951; Gordon, 1956).

In patients reported by Kern (1945), Singh and Jolly (1962) and others, absent or diminished cardiac pulsation on screening of the chest provided additional evidence of a pericardial effusion.

Radiographs in one of Buri's patients showed a pneumopyopericardium suggesting a pulmonary pericardial communication (Buri et al., 1955). Carter and Kerenes (1950) injected air into the pericardial sac and stated that the pneumopericardiograms obtained demonstrated a thickened pericardial layer and showed no evidence of loculation of the pericardial effusion.

Of great diagnostic importance may be the radiological changes produced by the amoebic liver abscess or abscesses associated with amoebic

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pericarditis. In the patient reported by Edwards (1947) there was a rounded localised elevation of the right diaphragm with overlying pulmonary changes. Screening of the chest in Singh's patient showed an immobile right diaphragm (Singh and Jolly, 1962). In Gordon's patient elevation of the diaphragm with an overlying pleural reaction was seen at the left base whilst radiographs of the abdomen showed posterior displacement of the gastric air bubble (Gordon, 1956). Kern (1945) stressed that in his patient the important radiological change of a bilaterally elevated diaphragm was not given proper consideration. In the patients reported by Coirault et al. (1955) and Norris and Beemer (1956) there were radiological changes of large left-sided effusions which were shown on aspiration to contain anchovy or chocolate coloured pus.

Alkan et al. (1961) noted that in addition to radiological changes in the chest suggesting subdiaphragmatic pathology a barium meal may be helpful in localising a left lobe abscess in that there may be displacement or indentation of the barium filled stomach by the abscess.

Delance (1960) reporting a patient with amoebic pericarditis claimed to have demonstrated evidence of hepatic adhesions by means of radiography following a pneumoperitoneum.

Attempts have been made to demonstrate hepatopericardial fistulas by means of radio-opaque substances. In 1936 Huard et al. outlined a left lobe abscess by injecting lipiodol. They were able

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to show that the abscess extended retrosternally but were unable to demonstrate an hepatopericardial communication.

Radiological findings in this study showed that in amoebic pericarditis changes in the cardiac outline consistent with a pericardial effusion are usual. In the early stage of the presuppurative lesion they may be absent (cases 52 and 54). In 2 patients with suppurative amoebic pericarditis changes in the cardiac silhouette similar to those seen with a left ventricular aneurysm were present (cases 57 and 58). In one patient (case 65) whose initial radiographs showed no evidence of a pericardial effusion these changes subsequently developed rapidly seen after admission.

In a few instances air I injected into the pericardium demonstrated thickening of the parietal layer and in one patient (case 59) lipiodol injected into the pericardium was subsequently seen to have drained into the liver demonstrating a hepatopericardial fistula.

Diagnostically the most helpful radiological features were those suggesting the presence of a liver abscess i.e. elevation or paresis of the left or right diaphragm. Radiological evidence of associated hepatic amoebiasis, however, is by no means invariable and in this study there were patients in whom I was unable to demonstrate such evidence.

Radiology was also of value in assessing the progress of the patient. Recovery was associated with return of the heart size and

/shape ...

shape to within normal limits and normal cardiac pulsation on screening. In others the decrease in heart size was associated with clinical signs of constrictive pericarditis and absent or decreased pulsation on screening.

PERICARDIAL AND LIVER ASPIRATION

Presuppurative Amoebic Pericarditis: In 1927 Piessinger and Casteran aspirated sterile serous fluid containing polymorphonuclear cells from the pericardium and sterile chocolate pus from an underlying liver abscess. Similar findings were reported by Stephan (1948). In one of the patients reported by Lamont and Peeler (1958) blood-stained fluid containing pus cells, lymphocytes and erythrocytes was aspirated initially and at necropsy pus was found in the pericardium indicating that the lesion had progressed from a presuppurative to a suppurative stage. The pericardium was not aspirated in patients considered to be suffering from presuppurative amoebic pericarditis reported by Purcell (1938), Edwards (1947), Cairault et al. (1955), Kelly et al. (1949). In the patients reported by Purcell (1938) and Kelly et al. (1949) pus was aspirated from a left lobe amoebic liver abscess.

Suppurative Amoebic Pericarditis: The available information concerning aspiration of the pericardial sac in patients with suppurative amoebic pericarditis reported during the past 20 years may be seen in table 21.

From his patient's pericardium, Kern (1945) aspirated several

/hundred ...

TABLE 21. THE DETAILS OF PERICARDIAL ASPIRATION IN 18 PATIENTS WITH SUPPURATIVE AMOEBIC PERICARDITIS REPORTED IN THE LITERATURE FROM 1945

N.R. = Not recorded.

Author	Character of Aspirate	Volume of 1st Aspirate (ml.)	Number of Aspirations	Total volume Aspirated (ml.)	Cells in Aspirate	Parasites Bacteria in Aspirate	Associated Aspirations
Kern 1945	Chocolate pus	600	Several	N.R.	R.B.C's Leucocytes 88 per cent polymorphs	Absent	Serous fluid from right pleura
Singh 1946	Anchovy sauce pus	40	2	70	N.R.	Absent	Liver abscess
Carter and Korones 1950	Pale orange pus	100	5 followed by surgical drainage	N.R.	Leucocytes R.B.C's	Absent	-
Buri et al. 1955 (3 patients)	No. 1 anchovy sauce pus	270 plus air	Several	N.R.	N.R.	E.histolytica detected	Left pleura Liver abscess
	No. 2 anchovy sauce pus	N.R.	6	N.R.	N.R.	Absent	Liver abscess
	No. 3 anchovy sauce pus	800	4	2,900	N.R.	Absent	None
Norris and Beemer 1956	Blood stained fluid	200	2	450	R.B.C's	N.R.	Left pleura pinkish pus
Gordon 1956	Anchovy sauce pus	600	2	650	R.B.C's Pus cells	Absent	Liver abscess
Hollender and Grenier 1957	Chocolate pus	N.R.	1 followed by surgical drainage 500 ml.	N.R.	N.R.	Absent	None
Pujol and de Preaumont (1957)	pus café au lait	400	N.R.	N.R.	Pus cells R.B.C's Charco-Leyden crystals	Absent	Surgical drainage of liver abscess
Scheffer 1958	Currant coloured pus	200	8	N.R.	N.R.	Absent	Left pleura
Parison et al. 1958	Chocolate pus	N.R.	1 followed by surgical drainage	N.R.	N.R.	E.histolytica detected	Left lobe liver abscess
Lamont and Pooler 1958 (Details of 2 patients) —	No. 4 pus	N.R.	Several followed by surgical drainage	2500	N.R.	N.R.	None
	No. 5 yellow pus	N.R.	Several followed by surgical drainage	2400	N.R.	N.R.	Surgical drainage liver abscess
Michon et al. 1959	Chocolate pus	150	2 followed by surgical drainage	500	N.R.	Absent	None
Alkan et al. 1961	Chocolate pus	600	Several	N.R.	N.R.	Absent	None
Singh and Jolly 1962	Anchovy sauce pus	N.R.	Several	6000	R.B.C's Pus cells	Staphylococcus	Pleura serous fluid
Delance 1960	Yellowish green pus Choclate	N.R.	3	N.R.	N.R.	Absent	None

hundred millilitres of chocolate coloured pus, which with subsequent aspirations changed to a brick red colour. Repeated direct examination and cultures showed no evidence of bacteria, acid-fast bacilli or parasites. Commenting in retrospect on this aspirate Kern noted that it corresponded to the characteristic description of the contents of an amoebic liver abscess and because of this the correct diagnosis should have been suspected.

Buri et al. (1955) were particularly successful when aspiration was attempted in their patients. They reported 3 patients, from each of whom they aspirated anchovy sauce pus from the pericardium. In 2 of these patients similar pus was aspirated from a left lobe liver abscess. From one of their patients large quantities of air were aspirated with the pericardial pus and E.histolytica identified in the pus. From another of their patients anchovy pus was aspirated from the left pleura.

With regard to the quantity of pus aspirated it can be seen from table 21 that up to 800 ml. (Buri et al., 1955) were obtained at the initial aspiration and that in Singh and Jolly's patient a total quantity of 6 litres was aspirated (Singh and Jolly, 1962). Kern (1945), Carter and Korones (1950), Alkan et al. (1961) found that repeated aspirations were required to relieve cardiac tamponade.

Anchovy sauce pus (Buri et al., 1955; Gordon, 1956) and chocolate coloured pus (Kern, 1945; Delance, 1960; Alkan, 1961) were the descriptive terms most frequently applied to the aspirate, but

/blood-stained ...

blood-stained fluid (Morris and Beemer, 1956) and yellow pus (Lamont and Pooler, 1958; Delanoe, 1960) were also aspirated.

In instances where the aspirate was examined varying quantities of red blood cells, pus cells or polymorphonuclear leucocytes were found (Kern, 1945; Carter and Korones, 1950; Gordon, 1956 and Singh and Jelly, 1962). The majority of aspirates showed no organisms or parasites on direct examination and were sterile on culture for bacteria and acid-fast bacilli. A significant feature was that in only 2 of the pericardial aspirates recorded in table 21 were E.histolytica identified (Buri et al., 1955; Farison et al., 1958).

In addition to the aspiration of pus from the pericardium pus was aspirated from the responsible left lobe liver abscess in 2 of the patients reported by Buri et al. (1955) and Farison et al. (1958). The responsible liver abscess was demonstrated at laparotomy in one of the patients reported by Lamont and Pooler (1958) and in the patient reported by Pujol and de Preaumont (1957).

In the present study, in only 2 of the 5 patients considered to be suffering from presuppurative amoebic pericarditis was it possible to establish the diagnosis by needle aspiration. Repeated aspiration showed that a further patient (case 65) had progressed from the pre-suppurative to the suppurative stage. In accord with the experience of previous workers the pericardial aspirates showed a predominance of red blood cells and neutrophils and the absence of organisms, acid-fast bacilli and E.histolytica.

The left lobe liver abscess responsible for the pericardial lesion was demonstrated by needle aspiration in 4 of the 5 patients with presuppurative amoebic pericarditis in this study. In the remaining patient it was demonstrated at laparotomy. In each of these 5 patients the pus aspirated from the liver was sterile on culture for bacteria and in 4 of the 5 it was of a typical anchovy sauce character. Trophozoites of E.histolytica were identified in the liver pus of 2 patients.

Pericardial aspiration was the single most important diagnostic procedure used in this study. By this method the suppurative nature of the pericardial effusion was established in 8 of the 10 patients with suppurative amoebic pericarditis. The findings on pericardial aspiration were similar in many respects to those of previous workers which have been mentioned above. In the majority of my patients repeated aspirations were required to relieve cardiac tamponade. The largest volume removed at a single aspiration was 1500 ml. and from the pericardium of one patient who required 10 aspirations a total of more than 6 litres of pus was removed. Findings of diagnostic significance were that the pus was of a typical anchovy sauce character in 6 of the 8 patients, was sterile on culture for bacteria in all but one patient and acid-fast bacilli were absent on direct examination and culture in each instance. The diagnosis was proved in 3 patients by the identification of E.histolytica in the pericardial pus and, in one of these patients, liver cells identified

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in the pericardial pus indicated the source of the effusion.

The safest and most convenient site for aspiration was found from experience to be a transdiaphragmatic approach through the left upper epigastrium in the angle between the xiphisternum and the costal margin.

The left lobe liver abscess responsible for the pericardial effusion was demonstrated by needle aspiration in 5 of the 10 patients with suppurative amoebic pericarditis in this study. In 4 of these 5 patients the pus aspirated from the liver was of a characteristic anchovy sauce colour and was sterile on culture for bacteria. Trophozoites of E.histolytica were identified in the liver pus from one patient.

ELECTROCARDIOGRAPHIC FINDINGS

Electrocardiographic changes compatible with pericarditis were seen in the patients reported by Carter and Koronos (1950), Gordon (1956), Lamont and Pooler (1958), and Alkan et al. (1961). In the majority of instances these changes provided additional evidence of pericardial involvement and were similar to electrocardiographic changes seen in other varieties of pericarditis (Friedberg, 1956). In the patient reported by Gordon (1956) recovery was associated with reversion of the electrocardiographic changes to normal.

The commonest electrocardiographic abnormality in this study was T-wave inversion in the standard leads, unipolar limb leads and

/chest ...

chest leads. The usual pattern of generalised S-T segment elevation was less frequently seen. In a few patients a low voltage pattern developed in association with the onset of constrictive pericarditis and in others recovery was associated with reversion of the T-waves to normal position.

The electrocardiographic tracings of normal healthy African males may show variations not seen or accepted in their European counterparts (Grusin, 1954; Brink, 1956; Powell, 1959). S-T segment elevation and T-wave inversion have been described amongst these changes and because of this difficulty may be experienced in interpreting the electrocardiographic changes in an African patient with a suspected pericardial effusion.

My experience with electrocardiography in amoebic pericarditis is that it plays a subsidiary role to the clinical and radiological findings in suggesting the diagnosis and that recovery is associated with return of the tracing to within normal limits.

EXAMINATION OF THE STOOLS

Laigret (1928) and D'Mello (1947) have reported the most unusual association of pericarditis with acute amoebic dysentery. Most patients reported to date with amoebic pericarditis have had no clinical or laboratory evidence of concomitant amoebic colitis. Exceptions have been those reported by Asar (1932), Melluy et al. (1949) and Berberian et al. (1951) in whose stools E.histolytica was

/identified ...

identified. Of interest is that Harts (1950), Buri et al. (1955) and Norris and Beemer (1956) found evidence of amoebic colitis at necropsy in their patients which had not been manifest or detected during life.

Clinical evidence of concomitant intestinal amoebiasis was present in only 2 of my patients with amoebic pericarditis. Two patients were similar to those reported by Buri et al. (1955) and others mentioned above in that necropsy revealed amoebic colitis which had not presented clinically or been detected during life. Because of this I feel that a diligent search should be made for E.histolytica in the stools of patients with suspected amoebic pericarditis.

CONCLUSIONS

Moderate to severe anaemia of a normochromic normocytic type which responds to treatment with emetine and chloroquine is frequently present in amoebic pericarditis. The white cell count in amoebic pericarditis is usually but not invariably raised. The sedimentation rate is always increased and recovery is accompanied by a decrease in the rate.

Radiologically the heart size and contour may be within normal limits in presuppurative amoebic pericarditis but changes compatible with a moderate to large pericardial effusion are usual. The majority of patients with suppurative amoebic pericarditis have radiological changes

/compatible ...

compatible with a moderate to large pericardial effusion but occasionally the alteration in cardiac contour may not be suggestive. Screening of the chest is helpful in the diagnosis and assessment of progress. Diagnostically the most important radiological changes in amoebic pericarditis are those produced by the responsible liver abscess. Injection of a contrast medium into the pericardium may demonstrate the thickness of the parietal pericardium or a hepatopericardial fistula. Such demonstrations are seldom essential for the diagnosis and should not be done as a routine.

Pericardial aspiration should be attempted in all patients with pericardial effusions of suspected amoebic origin. The aspiration of serous or serosanguineous fluid from the pericardium of a patient with the clinical features of a liver abscess should suggest the possibility of presuppurative amoebic pericarditis. The aspiration of sterile, anchovy sauce or chocolate sauce pus from a left lobe liver abscess in such a patient provides further evidence for the diagnosis, whilst the identification of E.histolytica in the liver pus is virtually diagnostic.

The aspiration of anchovy sauce or chocolate sauce pus from the pericardium should suggest the diagnosis of suppurative amoebic pericarditis. The identification of E.histolytica in this pus proves the diagnosis but failure to find the parasite in no way excludes the diagnosis. In any patient from whom pus is aspirated from the pericardium which is sterile on culture for bacteria and acid-fast bacilli,

/amoebic ...

amoebic pericarditis should be considered amongst the diagnostic possibilities. When pus is aspirated from a liver abscess in a patient with suppurative pericarditis an amoebic origin should be suspected and further evidence is provided when this pus is sterile on culture or is of an anchovy sauce character. The identification of E.histolytica in the liver pus proves the amoebic origin of the liver abscess and is very suggestive that the pericarditis is likewise amoebic in nature.

The electrocardiographic changes in amoebic pericarditis are similar to those reported in other varieties of pericarditis. Changes similar to those found in amoebic pericarditis may occur in the electrocardiograms of normal African males, and because of this, difficulty may be experienced in the interpretation of changes in African patients with suspected amoebic pericarditis. With recovery the electrocardiographic changes in amoebic pericarditis revert to normal but with the onset of constrictive amoebic pericarditis a low-voltage pattern may develop.

In patients with amoebic pericarditis, dysenteric stools are uncommon and E.histolytica seldom found. However, as present and previous necropsy findings have, in some cases, revealed colonic ulcers, the stools of patients with amoebic pericarditis should be frequently examined for E.histolytica.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF PERICARDIAL AMOEBIASIS

Michon et al. (1959) considered it significant that their patient had resided in an area where amoebiasis was endemic. Others have noted that a past history of dysentery may be important in that it suggests the possibility of pericardial amoebiasis (Coirault et al., 1955), but Carter and Koronos (1950) emphasise that a negative history is by no means significant since half to two-thirds of all patients with hepatic amoebiasis give no history of antecedent diarrhoea.

PRESUPPURATIVE AMOEBIC PERICARDITIS

Carter and Koronos (1950) in their review of the literature noted that in the patients reported with non-suppurative pericarditis the diagnosis was established either, by finding serous fluid in the pericardial sac at postmortem examination, or by appearance during life of a pericardial friction rub that disappeared after treatment with emetine hydrochloride.

The diagnosis in the patient reported by Laigret (1928) was based on the rather flimsy evidence of the association of a pericardial effusion which responded slowly to emetine and an acute dysenteric episode. In Purcell's patient, although the nature of the pericardial effusion was not established, the existence of a left lobe liver abscess was proved by needle aspiration of the liver and there was a satisfactory response to emetine (Purcell, 1938). Laha (1946) noted in his patient that the clinical picture coupled with the response to emetine established

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the case as one of amoebic hepatitis and associated dry pericarditis. The diagnosis was suggested in the patient described by Edwards (1947) by the clinical and radiological features of an associated liver abscess with a hepatobronchial fistula and a satisfactory response to emetine therapy.

Recently Lamont and Pooler (1958) reported a patient with a "sympathetic" pericardial effusion resulting from an adjacent liver abscess localised by needle aspiration. The nature of the pericardial effusion was not established but there was a satisfactory response to treatment with emetine and chloroquine.

Commenting on the clinical presentation in their patient Takaro and Bond (1958) stated that as the pericardial effusion was not aspirated they were unable to state whether cardiac enlargement was due to a frank suppurative process in the pericardial cavity or to a pericardial effusion secondary to the subphrenic process.

Considering the case reports mentioned above it is evident that the diagnosis of presuppurative amoebic pericarditis was largely dependent on the clinical presentation and response to emetine. In no instance was E.histolytica found or the nature of the pericardial fluid determined.

The diagnosis of presuppurative amoebic pericarditis in the present study was based on the clinical presentation, supportive radiological changes and the findings on pericardial and liver aspiration. The important role of pericardial and liver aspiration in the diagnosis

/has ...

has been previously stressed. Ideally to establish the diagnosis of a presuppurative amoebic pericardial effusion needle aspiration should yield pus containing E.histolytica from the responsible liver abscess and serous or serosanguineous fluid from the pericardium. That this ideal is seldom realised can be seen from the above reports and the findings in this study.

The diagnosis of a left lobe liver abscess was suggested by the clinical findings in each of my 5 patients with presuppurative amoebic pericarditis. In 4 of these patients the responsible liver abscess was localised by needle aspiration and in the remaining patient it was demonstrated at laparotomy. The grounds on which these liver abscesses were considered to be of amoebic origin have been previously mentioned and it was noted that the identification of E.histolytica in the liver pus proved the diagnosis in 2 of these patients.

The presuppurative nature of the pericardial effusion was proved by pericardial aspiration in 2 of my patients. In a further instance the diagnosis was based on the association of a pericardial friction rub with the left lobe liver abscess. In the 2 remaining patients the diagnosis of presuppurative rather than suppurative pericarditis was suggested by the clinical course, the response to treatment and the fact that pericardial drainage was not required in the management.

SUPPURATIVE AMOEBIC PERICARDITIS

Table 22 records the findings of Carter and Koremes who reviewed the literature in 1950 and claimed that in only 2 patients reported with suppurative amoebic pericarditis was the diagnosis established during life. From table 22 it can be seen that one of these was the patient reported by Huard and Meyer-May (1936) who were able to aspirate chocolate sauce pus from the pericardium and from a left lobe liver abscess. The other was the patient reported by Singh (1946) who made a tentative diagnosis of a pericardial effusion with an abscess of the liver from the clinical and radiological findings and was able to aspirate sterile anchovy sauce pus from both the pericardium and the responsible liver abscess. Lasage and Melnotte (1928), Berman (1946), Davis (1948) and Shaw (1949) describe 4 further instances prior to 1950, not mentioned by Carter and Koremes (1950), in which the diagnosis of suppurative amoebic pericarditis was established during life.

Kern (1945) in an admirable and critical assessment of his own clinical approach noted in retrospect that in his patient the symptomatology, radiological changes of a bilaterally elevated diaphragm and the character of the pericardial aspirate should have directed attention to the correct diagnosis.

In the literature since 1950 I have been able to find a further 20 patients with suppurative and 3 with presuppurative amoebic pericarditis. The details of these reports have been briefly summarised

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TABLE 22. THE DATA IN 58 CASES OF AMOEBIC PERICARDITIS AS REVIEWED BY CARTER AND KONONEN (1950)

AUTHOR	YEAR	NO. OF CASES	CASES OF PERICARDITIS		DIAGNOSIS DURING LIFE	RESULT	
			Suppurative	Non Suppurative		Recovery	Death
Howard and Hoover	1897	1	1		No	0	1
Craig	1924	12	2	10	No	0	12
Justi	1925	19	19		*	*	*
Surbek	1928	1	1		No	0	1
Laigret	1928	1		1	Yes	1	0
Katarepule	1932	1	1		No	0	1
Verges and Hermenjat-Gerin	1932	13	13		*	0	13
Huard and Meyer-May	1936	3	3		Yes (1 case)	1	2
Purcell	1938	1		1	Yes	1	0
Oehsner and DeBakey	1943	1	1		No	0	1
Herrilich	1943	1	1		No	0	1
Kern	1945	1	1		No	0	1
Laha	1946	1		1	Yes	1	0
Singh	1946	1	1		Yes	0	1
Edwards	1947	1		1	Yes	1	0

* Information not available.

in table 23. It can be seen that in 16 of these patients with suppurative amoebic pericarditis the diagnosis was made during life, a considerable advance on the findings of Carter and Koronas (1950) in their review of the literature prior to 1950.

Buri et al. (1955) made the diagnosis of suppurative amoebic pericarditis in 3 patients. They were able to aspirate anchovy sauce pus from the pericardium of each patient and from the responsible liver abscess in 2 patients. E.histolytica was identified in the pericardial aspirate of one patient and from the liver pus of another.

Gordon (1956) proved the existence of a left lobe liver abscess in his patient by needle aspiration. This abscess subsequently ruptured into the pericardium and anchovy sauce pus was obtained at pericardial aspiration.

Lament and Pooler (1958) established the diagnosis in 4 out of 5 of their patients with suppurative amoebic pericarditis. Each of these patients presented with a pericardial effusion and needle aspiration of the pericardium and liver and in some instances laparotomy, played an important role in diagnosis.

In the patient reported by Hollender and Grenier (1957) a provisional diagnosis of a left lobe liver abscess was made on the basis of clinical and radiological findings whilst subsequent rupture into the pericardium was suggested by the sudden development of a pericardial effusion and confirmed by the aspiration of chocolate coloured pus.

TABLE 23 THE DATA IN 23 PATIENTS WITH AMOEBIC PERICARDITIS REPORTED IN THE LITERATURE AFTER 1950

AUTHOR	YEAR	NO. OF CASES	CASES OF PERICARDITIS		DIAGNOSIS DURING LIFE	RESULT	
			Suppurative	Non Suppurative		Recovery	Death
Harts	1950	1	1		No		1
Buri et al.	1955	3	3		Yes(3)	1	2
Coirault et al.	1955	1		1	Yes	1	
Norris and Beemer 7	1956	1	1		No		1
Gordon	1956	1	1		Yes	1	
El Din et al.	1956	1	1		No		1
Hollender and Grenier	1957	1	1		Yes	1	
Pujol and de Preaument	1957	1	1		Yes	1	
Scheffer	1958	1	1		Yes		1
Farison et al.	1958	1	1		Yes	1	
Lamont and Peeler	1958	7	5	2	Yes(6)*	2**	5
Michon et al.	1959	1	1		Yes		1
Delance	1960	1	1		Yes	1	
Alkan et al.	1961	1	1		Yes		1
Singh and Jolly	1962	1	1		Yes	1	

* The diagnosis was established during life in 4 of the 5 suppurative cases and in the 2 non-suppurative cases.

** Each of these was a case of non-suppurative pericarditis.

The reports by Pujol and de Preaumont (1957), Scheffer (1958), Farison et al. (1958), Michon et al. (1959), Delance (1960), Alkan et al. (1961) and Singh and Jolly (1962) were of patients who in each instance developed a pericardial effusion with signs of tamponade. The diagnosis in these patients was based in most instances on the aspiration of either chocolate pus or anchovy sauce pus from the pericardium.

As has been previously mentioned it is significant that of the 20 patients reported from 1950 with suppurative amoebic pericarditis E.histolytica has only been identified in the pericardial aspirate of 2 (Buri et al., 1955; Farison et al., 1958).

Takaro and Bond (1958) stated, "Pericardial amebiasis is proved by the aspiration of "anchovy-sauce" pus from the pericardial sac from which Entamoeba histolytica can be isolated, or it may be inferred in the presence of symptoms and signs of pericardial effusion in a patient with a proved amebic hepatic abscess or pleuropulmonary amebiasis."

The diagnosis was established soon after hospital admission in 8 of the 10 patients with suppurative amoebic pericarditis reported in this study. In the remaining 2 the clinical picture was atypical and the liver abscess and the suppurative pericarditis were only demonstrated at necropsy.

The diagnosis was suggested in the majority of my patients by the clinical association of symptoms and signs compatible with a left lobe liver abscess and those of a pericardial effusion with severe

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cardiac tamponade. In a few severe retrosternal pain and signs of peripheral vascular collapse dominated the clinical picture and radiologically the enlarged cardiac shadow with a globular outline suggested the possibility of a pericardial effusion. Fortunately myocardial infarction is a rare occurrence in African patients attending this hospital and it was seldom that this possibility required consideration. Occasionally the rapidity with which the pericardial effusion and cardiac tamponade developed suggested the diagnosis.

Pus was aspirated from the pericardium in each of the 8 patients in whom the diagnosis was established and in 5 of these patients pus aspirated from a left lobe liver abscess demonstrated the likely source of the pericardial pus. The findings on examination of the pericardial and liver aspiration which suggested an amoebic origin have been previously recorded. My experience was similar to that of workers mentioned above in that the concomitant aspiration of pus from a liver abscess and the pericardium and the anchovy sauce colour of the pus were major factors in establishing the diagnosis. In 3 of my patients the diagnosis was proved by the identification of E.histolytica in the pericardial pus, a finding rarely reported in the literature. In one of my patients liver cells identified in the pericardial pus left no doubt as to its origin. As far as I can ascertain this is the first report of such a finding in amoebic pericarditis.

DIFFERENTIAL DIAGNOSIS

Tuberculous pericarditis was the favoured diagnosis throughout the clinical course in the patient reported by Kern (1945) and it was only at necropsy that the amoebic origin was demonstrated. Gordon (1956) stated that when the diagnosis of liver abscess is not apparent, a serous or sympathetic effusion may easily be confused with pericarditis from other causes, particularly tuberculosis. The truth of this statement was demonstrated by the experience of Lamont and Peeler (1958). Blood-stained serous fluid was aspirated from a pericardial effusion in one of their patients and treatment started with streptomycin and isoniazid; necropsy revealed a suppurative amoebic pericarditis. Other reports in which tuberculous pericarditis featured prominently amongst the diagnostic possibilities have been documented by Coirault et al. (1955), Norris and Beemer (1956), Scheffer (1958) and Delance (1960).

Huard et al. (1933) reported a patient in whom necropsy revealed liver abscesses, pulmonary abscesses and a pericardial effusion. They favoured a bacterial origin but as the pus initially drained from the liver abscesses was sterile on culture for bacteria they were not prepared to discard a possible amoebic etiology. Judging from the order in which chemotherapy was administered in the treatment of the patient reported by Delance (1960) a diagnosis of pyogenic pericarditis was favoured initially, followed by tuberculous pericarditis and finally the correct diagnosis was established.

Stephan (1948) mistook the retrosternal pain and the cardiovascular findings in one of his patients to be those of a coronary thrombosis. Sudden circulatory collapse and the ensuing signs led to a diagnosis of a thrombotic process in the aorta with embolisation of the left femoral artery in the patient reported by Hartz (1950). In the patient reported by Ceirault et al. (1955) tuberculous pericarditis, rheumatic pericarditis and subacute bacterial endocarditis were considered amongst the diagnostic possibilities.

Schrire (1959) stated that pericarditis is one of the commonest cardiovascular diseases in the Bantu. He found that tuberculosis was by far the commonest cause and that the peak incidence was in Bantu males in the fourth and fifth decades. Tuberculosis is the commonest cause of pericarditis in African (Bantu) patients seen at this hospital, amoebic pericarditis and other varieties of pericarditis being rare in comparison. Because of this tuberculous pericarditis featured prominently in the differential diagnosis of my patients. Fortunately, it was possible in the majority of instances to reach a fairly conclusive diagnosis of amoebic pericarditis by demonstrating the responsible liver abscess, or from the examination of the pericardial aspirate. Patients in whom the signs of a liver abscess are not apparent or are equivocal present the greatest difficulty in diagnosis. There was one such patient in this study (case 53) in whom initially tuberculous pericarditis could not be excluded but in whom subsequently the responsible

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left lobe amoebic liver abscess was demonstrated. If there are any grounds for suspecting an amoebic origin in a patient with pericarditis of undetermined origin every effort should be made to demonstrate the responsible liver abscess. Screening of the left diaphragm, barium meal and pneumoperitoneum may be helpful in this respect and in some instances exploratory aspiration of the liver or a diagnostic laparotomy may be required.

Schrire (1959) noted that pyogenic pericarditis appears to be rare in the Bantu. In my experience the condition is rare in African (Bantu) patients at this hospital and in the few examples I have seen has resulted by spread from an adjacent suppurative focus in the chest, as part of a generalised septicaemia state or as the result of secondary infection of an amoebic pericarditis. I have never seen a patient in whom a pyogenic abscess of the liver has ruptured into the pericardium, a sequence reported in a child by Outerbridge and Sun (1951). The distinction between amoebic pericarditis and pyogenic pericarditis may be difficult and pus aspirated from the pericardium should always be examined and cultured for pyogenic organisms. The isolation of such organisms, however, does not exclude the possibility of a secondarily infected amoebic pericarditis.

The experience of Stephan (1948) previously mentioned indicates that the clinical presentation of amoebic pericarditis may simulate that of coronary thrombosis. Fortunately ischaemic heart disease and myocardial infarction are very uncommon in African patients attending

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this hospital and were not a problem in the differential diagnosis of my patients.

There was one patient seen during the course of this study with a blood-stained effusion in whom amoebic pericarditis was considered amongst the diagnostic possibilities but necropsy revealed a malignant pericarditis. It is conceivable that the presuppurative presentation of amoebic pericarditis in patients in whom the responsible liver abscess is not apparent could be mistaken for a viral or rheumatic pericarditis.

The condition of cardiac or myocardial failure of undetermined etiology is commonly encountered in African patients attending this hospital. It seldom presented a problem in differential diagnosis in this study.

CONCLUSIONS

In any patient presenting with pain or guarding and tenderness of undetermined origin which is localised or maximal in the epigastrium a left lobe liver abscess should be suspected. When such an abscess points anteriorly or occupies an anterior position it can usually be demonstrated by needle aspiration. If the abscess occupies a posterior position radiological investigations such as screening of the chest, a barium meal or pneumoperitoneum may assist in its localisation and a laparotomy may be required for its demonstration.

In any patient with a proved or suspected left lobe liver abscess a careful and repeated search should be made for signs of

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pericardial involvement. The diagnosis of presuppurative pericarditis may be inferred when a pericardial friction rub is heard in a patient with a left lobe liver abscess. It is confirmed by the aspiration of serous or seresanguineous fluid from the pericardium and amoebic pus from the responsible liver abscess.

The diagnosis of suppurative amoebic pericarditis should be suspected in any patient with a suspected or proved liver abscess who develops severe retrosternal pain and signs of circulatory collapse or signs of a pericardial effusion and cardiac tamponade.

It should be suspected in any patient from whom sterile pus is aspirated from the pericardium and is likely when this pus is of an anchovy or chocolate sauce colour. The diagnosis of suppurative amoebic pericarditis is confirmed by the identification of E.histolytica in the pericardial or liver pus.

In reported presentations in which signs of the responsible liver abscess have not been apparent amoebic pericarditis has been most frequently mistaken for tuberculous pericarditis. Suppurative amoebic pericarditis may be confused with a pyogenic pericarditis and in a patient with a pyogenic pericarditis the possibility of a primary amoebic etiology should not be overlooked. Ischaemic heart disease and other varieties of pericarditis may require consideration in the differential diagnosis of amoebic pericarditis.

THE MANAGEMENT OF PERICARDIAL AMOEBIASIS

PRESUPPURATIVE AMOEBIC PERICARDITIS

The administration of emetine hydrochloride and aspiration of the responsible liver abscess formed the basis of treatment in the patient reported by Purcell (1938). The satisfactory management, along similar lines, of presuppurative amoebic pericarditis has also been reported by Piessinger and Casteran (1927), Stephan (1948) and Lamont and Pooler (1958).

As the patient reported by Edwards (1947) coughed up the contents of the responsible liver abscess through a hepatobronchial fistula, liver aspiration was not necessary and there was a satisfactory response to emetine hydrochloride.

Recovery in the patient reported by Coirault et al. (1955) followed the administration of emetine hydrochloride and surgical drainage of the amoebic liver abscess and associated amoebic empyema.

The management of presuppurative amoebic pericarditis in this study was essentially similar to the regime used by Piessinger and Casteran (1927), Purcell (1938), Stephan (1948) and others mentioned above in that treatment was directed primarily at the responsible liver abscess.

Each of my patients received emetine and chloroquine and in some instances repeated courses were required. Drainage of the responsible liver abscess was accomplished by needle aspiration in 3 of the 5 patients in this group and in the 2 remaining patients a laparotomy was required.

Presuppurative amoebic pericarditis may progress suddenly to the more serious and often fatal suppurative stage. Early and adequate drainage of the responsible liver abscess is therefore essential. If needle aspiration of the liver abscess is unsuccessful or yields unsatisfactory quantities of pus laparotomy should be performed to permit adequate drainage.

SUPPURATIVE AMOEBIC PERICARDITIS

Chemotherapy: The drug emetine hydrochloride formed the basis of treatment in 3 of the 4 recoveries from suppurative amoebic pericarditis encountered before 1950 (Huard et al., 1936; Berman, 1946; Shaw, 1949). This preparation was however also used in the fatal cases reported by Scuderi (1928), Huard et al. (1933) and Singh (1946).

A glance at table 24 will show that emetine was used in the treatment of 7 of the 8 recoveries reported in the literature after 1950. The exception was one of the patients reported by Buri et al. (1955) who was treated with Aralen (chloroquine).

Gordon (1956), Lamont and Pooler (1958) and Singh and Jolly (1962) used chloroquine in addition to emetine in the treatment of their patients, whilst Scheffer (1958) and Delanoe (1960) used stovarsal and emetine.

Emetine hydrochloride and chloroquine (sulphate or phosphate) are the two most potent tissue amoebicides available (Wilmot, 1962). All patients with amoebic pericarditis in this study received both

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TABLE 24. DRAINAGE PROCEDURES AND AMOEBICIDES USED IN 25 PATIENTS WITH SUPPURATIVE AMOEBIC PERICARDITIS REPORTED IN THE LITERATURE

P = Aspiration the only form of direct pericardial drainage

AUTHOR	YEAR	PERICARDIAL ASPIRATION	LIVER ABSCESS ASPIRATION	SURGICAL DRAINAGE PERICARDIUM	SURGICAL DRAINAGE LIVER ABSCESS	AMOEBICIDES	RECOVERY
Huard and Meyer-May	1935	+	-	-	+	Emetine Stovarsol	Yes
Kern	1945	+P	-	-	-	None	No
Berman	1946	+P	-	-	-	Emetine	Yes
Singh	1946	+P	+	-	-	Emetine	No
Shaw	1948	?	-	-	+	Emetine	Yes
Davis	1948	?	-	+	+ (via pericardium)		Yes
Carter and Korones	1950	+	-	+	+	Emetine	No
Buri et al.	1955(1)	+P	+	-	-	?	No
	(2)	+P	+	-	-	Aralen	Yes
	(3)	+P	-	-	-	?	No
Norris and Beemer	1956	+P	-	-	-	None	No
Gordon	1956	+P	+	-	-	Emetine Chloroquine	Yes
Hollender and Grenier	1957	+	-	+	+	Emetine	Yes
Pujol and de Preaumont	1957	+	-	-	+	Emetine	Yes
Scheffer	1958	+P	-	-	-	Emetine Stovarsol	No
Parizon et al.	1958	+	-	-	+	Emetine	Yes
Lamont and Pooler	1958(1)	+P	-	-	-	None	No
Cases 2 & 6 presuppurative amoebic pericarditis	(3)	-	+	+	+	Emetine Chloroquine	No
Case 7 no details	(4)	+	-	+	-	Emetine Chloroquine	No
	(5)	+P	-	-	+	Emetine Chloroquine	No
Michon et al.	1959	+P	-	-	+	Emetine	No
Delanoe	1960	+	-	-	-	Emetine Stovarsol	Yes
Alkan et al.	1961	+P	-	-	-	Emetine	No
Singh and Jolly	1962	+P	-	-	-	Emetine Chloroquine	Yes
Wilmot	1962	+	-	+	-	Emetine	Yes

preparations in the usual doses and, when judged necessary, further courses were administered.

A wide range of antibiotics have been employed in the treatment of suppurative amoebic pericarditis. In some instances they have been administered prior to establishing the diagnosis and when either a tuberculous or pyogenic etiology was suspected (Coirault et al., 1955; Scheffer, 1958; Delance, 1960). The use of antibiotics without tissue amoebicides invariably resulted in failure. Others have given antibiotics in conjunction with tissue amoebicides even when the pericardial aspirate was sterile on culture (Gerden, 1956; Hollender and Grenier, 1957; Pujol and de Preaumont, 1957). Hollender and Grenier (1957) injected aureomycin into the responsible liver abscess and Michon et al. (1959) introduced penicillin, streptomycin and hydrocortisone into the pericardial sac. Takaro and Bond (1958) irrigated the liver abscess in their patient with solutions of emetine and streptokinase-streptodornase.

Most of my patients received antibiotics in addition to tissue amoebicides. In 2 instances secondary bacterial invasion necessitated their use, in the remaining patients they were judged necessary because of the danger of bacterial contamination from repeated pericardial aspiration. I have noted previously in this study that it is questionable whether antibiotics are of value in the prophylaxis of secondary bacterial invasion of amoebic foci. Their value when used for this purpose in my patients could not be assessed. Secondary bacterial invasion may

/necessitate ...

necessitate the injection of antibiotics into the pericardium. The above exception excluded, it was my policy to avoid the intra-pericardial injection of drugs, air or contrast media in my patients.

Scuderi (1928), Singh (1946) and Michon et al. (1959) used digitalis preparations in the management of their patients. It was difficult to decide whether true cardiac failure was present in addition to cardiac tamponade in my patients and whether rightly or wrongly, digitalis and diuretics were used in most instances.

Drainage Procedures: It has been stated that critical tamponade is rare in medical cases and that in practice it is rarely necessary to tap a pericardial effusion (Wood, 1956). This statement is certainly not applicable to suppurative amoebic pericardial effusions as Kern (1945), Carter and Kerenes (1950), Alkan et al. (1961) and others found that repeated aspirations were necessary in their patients to relieve or prevent severe tamponade.

Table 24 briefly summarises the details of the drainage procedures employed in 25 patients reported in the literature with suppurative amoebic pericarditis. Depending on the type of procedure used these patients may be divided into two groups.

The first includes those in whom needle aspiration of the pericardium was used with or without indirect drainage of the pericardium via needle aspiration of the responsible liver abscess (Kern, 1945; Singh, 1946; Gordon, 1956; Singh and Jolly, 1962).

Needle aspiration of only the pericardial effusion was carried out with satisfactory recoveries in the patients reported by Delance (1960) and Singh and Jolly (1962). In the recoveries reported by Buri et al. (1955) and Gordon (1956) both the pericardial effusion and the responsible liver abscesses were aspirated.

The second group includes patients in whom more radical surgical drainage was employed, either directly via a thoracotomy and pericardiotomy, or indirectly via laparotomy and drainage of the responsible liver abscess. (Provided the hepatopericardial fistula is patent drainage of the pericardial sac via the liver abscess should be possible.) An example of direct pericardial drainage was quoted by Davis (1948) in which the pericardium and left lobe liver abscess were drained by a single tube. This patient recovered and is known to have survived for 11 years. Hollender and Grenier (1957) successfully drained the pericardial effusion in their patient through a thoracotomy and subsequently performed a laparotomy to drain a left lobe liver abscess. Wilmot (1962) claimed personal knowledge of a patient subjected to surgical drainage of the pericardium in whom secondary infection was the main obstacle to recovery which, however, was eventually complete. The patients reported by Carter and Korones (1950) and Lamont and Pooler (1958) whose pericardial effusions were drained surgically did not survive. From a personal knowledge of Lamont and Pooler's patients it can be stated that secondary bacterial invasion invariably followed surgical drainage and in 3 of these patients constrictive pericarditis was found at

/necropsy ...

necropsy.

Huard and Meyer-May (1935) reported the first recovery from suppurative amoebic pericarditis. This followed emetine treatment and surgical drainage of the left lobe liver abscess. It is worth noting that these workers were unable to demonstrate a hepatopericardial fistula either radiologically by injection of contrast into the liver abscess or at surgery. Shaw (1949), Pujol and de Preaumont (1957) and Parison et al. (1958) have each reported recovery from suppurative amoebic pericarditis following surgical drainage of the responsible liver abscess.

Drummond and Lamont (1959) discussing drainage procedures in amoebic pericarditis stated that adequate drainage in the suppurative phase is provided by an indwelling polythene tube in the posterior pericardium. They felt that simple aspiration is probably inadequate if later possible constrictive pericarditis is to be avoided. Fiegel (1959) considered that rupture of an amoebic liver abscess into the pericardium required urgent surgical intervention. In her opinion not to operate was to expose the patient to grave peril. She favoured laparotomy with drainage of the pericardium via the liver abscess rather than direct drainage via a thoracotomy.

Needle aspiration of the pericardium was the drainage procedure employed in my patients. The reasons why this was used in preference to surgical drainage are as follows. Firstly, the

/majority ...

majority of my patients were suffering from severe tamponade on admission in addition to the systemic effects of long standing hepatic amoebiasis. To subject such a patient to the added stress of a general anaesthetic and a major surgical procedure was considered a hazardous undertaking. It is irrational to subject a critically ill patient to major surgery before attempting the equally effective and much less traumatic alternative of drainage by needle aspiration. Secondly, my experience of surgical drainage of amoebic liver abscess, amoebic empyema and amoebic pericarditis at this hospital has been that secondary bacterial invasion invariably results, with increased morbidity, if not mortality. It is noteworthy that Singh (1946) discussing his preference for needle aspiration as opposed to surgical drainage mentioned the above factors and reached the same conclusions.

In 5 of my patients with suppurative amoebic pericarditis needle aspiration was used to drain the source of the pericardial effusion which was a liver abscess of the left lobe. In instances where an abscess of the liver was diagnosed on clinical grounds the procedure practised was as follows. Firstly localisation of the liver abscess was attempted by needle aspiration. Having determined the existence of an abscess as much pus was drained as possible, and needle aspiration of the pericardium was then attempted. When more than 200 ml. of pus was drained from the liver abscess on initial aspiration further aspirations were attempted at 2 to 3 day intervals.

It can be seen from table 24 that there have been only 3

/reported ...

reported recoveries following direct surgical drainage of the pericardium in suppurative amoebic pericarditis (Davis, 1948; Hollander and Grenier, 1957; Wilmot, 1962). Vergos and Hermenjat-Gerin (1932) suggested that if a left lobe liver abscess be found to have extended into the pericardium at laparotomy the incision should be extended superiorly, the sternum split and the pericardium exposed and drained. There have been no instances reported in which this procedure has been attempted. (Huard and Meyer-May (1936) used this incision to drain a liver abscess.) As has been mentioned previously the poor general condition of my patients precluded such major surgical procedures.

Reviewing the literature I noted there had been 4 reported recoveries following surgical drainage of the responsible liver abscess (Huard and Meyer-May, 1935; Shaw, 1949; Pujol and de Preaumont, 1957; Farison et al., 1958). Provided it can be shown at surgery that the hepatopericardial fistula is patent and drainage of the liver abscess relieves tamponade this form of drainage would appear to be more satisfactory than direct drainage of the pericardium via a thoracotomy. The reasons for this are as follows: firstly, the patient is subjected to a laparotomy rather than a thoracotomy; secondly, the pericardium can be drained at the most dependent site, and finally, both the liver abscess and the pericardial effusion can be drained through the same incision.

In this study surgical drainage was attempted in only one instance (case 59) when repeated needle aspiration of the pericardium failed to relieve cardiac tamponade. A polythene tube was introduced into the pericardial cavity but proved unsatisfactory as a vehicle for

/drainage ...

drainage.

CONSTRICTIVE AMOEBIC PERICARDITIS

It has been mentioned previously in this work that patients with suppurative amoebic pericardial effusion may progress to develop constrictive pericarditis. Lamont and Pooler (1958) noted that the phase of constrictive pericarditis in those who survive intrapericardial rupture may take weeks or even months to develop. They stated, "it seems likely that a carefully timed pericardectomy would be the only possible therapy for this apparently inevitable sequel." Wilmot (1962) quoted Schrire as having seen a patient in whom the diagnosis was presumptive and who developed "acute severe (pericardial) constriction" which required surgery and was followed by recovery. If indeed this patient did have a constrictive amoebic pericarditis Schrire has seen the first patient to have recovered from this condition.

Three of my patients developed constrictive pericarditis. Two died shortly after attempted pericardectomy whilst the remaining patient survived a pericardectomy to make a complete recovery.

CONCLUSIONS

Both emetine hydrochloride and chloroquine should be used in the treatment of amoebic pericarditis. In some instances more than one course of treatment with these preparations may be required.

When bacterial contaminants are grown on culture of the

/pericardial ...

pericardial or liver aspirate the appropriate antibiotics should be given and solutions of these injected locally into the liver abscess or pericardium. When repeated pericardial aspirations are required the use of an antibiotic prophylactically to prevent secondary bacterial contamination is questionable.

In presuppurative amoebic pericarditis early and adequate drainage of the responsible liver abscess is essential to prevent progress to the suppurative state. Needle aspiration is the method of choice and should be repeated at 48 to 72 hour intervals until less than 100 ml. of pus is obtained with successive aspirations. Should needle aspiration of the responsible liver abscess be unsuccessful or provide inadequate drainage a laparotomy should be performed to locate and drain the abscess.

Immediate drainage of the pericardium is essential in suppurative amoebic pericarditis to relieve cardiac tamponade. Needle aspiration using a trans-diaphragmatic approach through the left xiphisternal-costal angle is the method of choice. A wide bore needle should be used and as much pus as possible removed at each aspiration. Aspirations should be repeated at 48 to 72 hour intervals or as cardiac tamponade necessitates until less than 100 ml. of pus is obtained at successive aspirations.

If, in suppurative amoebic pericarditis, needle aspiration of the pericardium is unsuccessful or fails to relieve cardiac tamponade a laparotomy should be performed, the responsible liver

/abscess ...

abscess drained and the hepatopericardial communication widened to provide adequate drainage for the pericardial effusion. If adequate drainage of the pericardial effusion cannot be provided at laparotomy this should be done via a thoracotomy.

Although there were patients in this study in whom aspiration of the liver abscess was not necessary aspiration drainage of the responsible liver abscess should probably be attempted on all patients with suppurative amoebic pericarditis.

Patients who recover from the acute episode should be carefully observed for symptoms and signs of constrictive pericarditis. If these develop provided they are not progressive the patient should if possible be observed for a period of at least 2 - 3 months before pericardiectomy is considered. In some instances clinical, radiological and electrocardiographic evidence of rapidly progressive constrictive pericarditis may necessitate an early pericardiectomy.

THE PROGNOSIS IN PERICARDIAL AMOEBIASIS

PRESUPPURATIVE AMOEBIC PERICARDITIS

Huard and Meyer-May (1936) stated that the prognosis in amoebic pericarditis of the serous and purulent aseptic types is dependent upon recognition and treatment of the responsible liver abscess. Recognition and treatment of the responsible liver abscess resulted in complete recovery in the patients with presuppurative amoebic pericarditis reported by Purcell (1938), Laha (1946), Edwards (1947), Stephan (1948), Coirault et al. (1955) and in 2 of those reported by Lamont and Pooler (1958). In a further patient reported by Lamont and Pooler (1958) the pericardial aspirate was compatible with a presuppurative pericarditis but necropsy showed a suppurative amoebic pericarditis. Failure to recognise and treat the responsible liver abscess and progress from the presuppurative to the suppurative stage in this patient were responsible for mortality.

In this study the 5 patients (cases 51 - 55) with presuppurative amoebic pericarditis recovered completely. The remaining patient (case 65) progressed to the suppurative stage and subsequently recovered. The excellent prognosis in my patients and in those reported in the literature mentioned above, was largely the result of early recognition, adequate drainage and treatment of the responsible liver abscess with emetine and/or chloroquine. Failure to establish the

/diagnosis ...

diagnosis at this stage may result in progression to the serious and often fatal suppurative state.

SUPPURATIVE AMOEBIC PERICARDITIS

Vergos and Hermenjat-Gerin (1932) quoted case reports by Rouis (1860) and Petridis (1920) whose patients died within minutes or hours of intra-pericardial rupture. Others who have reported rapid death following rupture have been Wilmot (1949) and Hartz (1951). Purcell (1938) stated that leakage from the liver abscess into the pericardium must be rapidly fatal.

Prior to 1950 there had been only 4 recoveries from suppurative amoebic pericarditis reported in the literature. These were the patients of Huard and Meyer-May (1935), Berman (1946) and those quoted by Davis (1948) and Shaw (1949). The patient quoted by Davis (1948) was known to have survived for 11 years without evidence of constriction.

In the literature since 1950 I have been able to find a further 8 survivors amongst 20 patients reported with suppurative amoebic pericarditis. The details of the management of these patients have been recorded in table 24. It is noteworthy that in the last 12 years there have been 8 survivors whereas in the first half of this century only 4 were reported. One of the reasons for this improvement in prognosis is undoubtedly the greater frequency with which the correct diagnosis has been established.

It can be seen from table 24 that 10 of the 12 survivors from

/suppurative ...

suppurative amoebic pericarditis were treated with emetine. Exceptions were, one of the patients reported by Buri et al. (1955) who was treated with aralen (chloroquine), and the patient quoted by Davis (1948) who received no emetine. To date, the patient quoted by Davis (1948) has been the only one to recover from suppurative amoebic pericarditis without being treated with the tissue amoebicides emetine or chloroquine.

Kern (1945) and Carter and Koromes (1950) showed that patients may survive for weeks without treatment with tissue amoebicides provided cardiac tamponade is relieved by needle aspiration or surgical drainage. Adequate drainage of the pericardial sac either directly or indirectly via the liver abscess was instituted in each of the 8 recoveries from suppurative amoebic pericarditis reported in the literature since 1950 (see table 24). To date, there have been no recoveries when drainage procedures have not been employed. In the acute stage death may be prevented by relief of cardiac tamponade, whilst the continued removal of pericardial fluid reduces morbidity and may decrease the likelihood of progression to a constrictive phase.

Early recognition and treatment was certainly a factor which influenced the prognosis in the 6 patients (60 per cent) who recovered from suppurative amoebic pericarditis in this study. In each of these patients, the correct diagnosis was established within hours of admission, cardiac tamponade was relieved, and treatment with emetine and chloroquine was commenced. For reasons previously mentioned I consider that drainage of the pericardium by needle aspiration rather than surgical incision

/improved ...

improved the prognosis in my patients.

Secondary bacterial invasion is known to worsen the prognosis of an amoebic liver abscess or amoebic empyema (Oehsner and DeBakey, 1936). There has been scant reference in the literature to its effect in amoebic pericarditis. Wilmot (1962) suggested that secondary bacterial infection may be a factor in the onset of constriction. This suggestion could well be applicable to the 3 patients reported by Lamont and Pooler (1958) and to 2 of my patients who developed constrictive pericarditis.

Wilmot (1962) stated, "One must conclude that constrictive pericarditis may follow amoebic pericarditis, but it is as yet uncertain whether secondary bacterial infection of the pus is a necessary factor for this to take place." Three patients in this study developed constrictive pericarditis, in 2 of these there was evidence of secondary bacterial infection but in the remaining patient there was no such evidence. Progression to a stage of constriction necessitated pericardiectomy which was responsible for the death of 2 of my patients. The surgeon concerned noted that in each of these patients greater difficulty was experienced in attempting to free the myocardium than was usual in patients with constrictive tuberculous pericarditis. The remaining patient with constrictive pericarditis made a complete recovery following pericardiectomy.

Each of the 5 patients with suppurative amoebic pericarditis and the one patient with constrictive amoebic pericarditis have been

/followed ...

followed for periods ranging from one to 2 years and have remained well with no evidence of constriction.

CONCLUSIONS

The prognosis in presuppurative amoebic pericarditis is dependent upon early recognition of the responsible liver abscess, adequate drainage of this abscess and treatment with emetine and chloroquine. If these criteria are fulfilled the prognosis in this type of amoebic pericarditis is excellent.

The recovery rate (60 per cent) from suppurative amoebic pericarditis in this study represents a notable improvement by comparison with that estimated from reports in the literature in the past 60 years. The factors responsible for this encouraging improvement in prognosis are considered to be the early recognition of the condition, the immediate and continued relief of cardiac tamponade by needle aspiration of the pericardium and the early institution of treatment with emetine and chloroquine.

Secondary bacterial invasion of the pericardial effusion worsens the prognosis in amoebic pericarditis and in some instances may be a factor in determining the onset of constrictive pericarditis. Constrictive amoebic pericarditis, however, can occur in the absence of secondary bacterial invasion of the pericardium.

The prognosis in suppurative amoebic pericarditis is worsened by progress to a stage of constrictive pericarditis.

SUMMARY

Sixty-five patients with thoracic amoebiasis have been studied, 50 of these presented with pleuropulmonary amoebiasis and 15 with pericardial amoebiasis.

The 50 patients with pleuropulmonary amoebiasis represent the largest personal series documented to date. The clinical and radiological findings, the relevant investigations and the details of management and progress have been recorded in these patients. Thirty presented with predominant pulmonary involvement and 20 with predominant pleural involvement. Combined lesions were found in 17.

The clinical presentation was fairly characteristic in over 90 per cent of these patients who had symptoms and signs referable to the right lower chest and right upper quadrant of the abdomen. Only 2 presented with signs at the left lung base.

The average duration of illness before admission was 9 to 10 weeks. Major symptoms were pain in the right lower chest and right upper quadrant of the abdomen associated with a cough productive of blood-stained or reddish-brown sputum and dyspnoea. Twelve per cent experienced dysenteric symptoms on admission and 26 per cent admitted to a past episode of dysentery.

Moderate pyrexia, evidence of weight loss, pale mucosa and clubbing of the fingers were features noted on general examination. Approximately half the patients with pulmonary lesions produced typical

/anchovy ...

anchovy sauce sputum. On local examination signs suggestive of consolidation, an elevated diaphragm or pleural effusion were found at the right lung base and in the majority there was guarding and tenderness in the right upper quadrant of the abdomen or a tender hepatomegaly. Thirty-seven patients were found to have a normocytic normochromic anaemia and in approximately half of these this was severe. Moderate elevation of the white cell count was usual and an increase in the sedimentation rate invariable.

On the basis of clinical and radiological findings each of the 30 patients in the pulmonary group was considered to have a hepatobronchial fistula. In 12 there was an associated abscess in the right lower lung, and in the majority of the remaining 18, pneumonic changes were seen in this situation. In 23 of the 30 patients with pulmonary lesions an elevated right diaphragm suggested the source of the lesion and in 9 this was established by the aspiration of pus from a liver abscess. The diagnosis was proved in 10 patients in this group by the identification of E.histolytica in the sputum.

The pleural group included 5 patients with presuppurative effusions and 15 with suppurative effusions. In 4 of those with suppurative effusions there was clinical and radiological evidence of a bronchopleural fistula. Pus was aspirated from the pleural cavity in 13 of the 15 patients with suppurative effusions, in 9 its typical anchovy sauce character suggested the diagnosis and in 6 the identification of E.histolytica in the pus proved the diagnosis. In 14 of the

/20 ...

20 patients with pleural amoebiasis an elevated right diaphragm suggested the source of the pleural effusion and in 5 the aspiration of pus from a liver abscess established this source.

Emetine and chloroquine formed the basis of therapy in the pleuropulmonary group and removal of pus from the pleura, liver and lung was done by needle aspiration and postural drainage. Drainage by surgical incision was rarely necessary.

Two patients with pulmonary and 4 patients with pleural amoebiasis died, resulting in a mortality for the group of 12 per cent. Twenty-eight patients with pulmonary and 16 with pleural amoebiasis made a complete recovery. Residual clinical and radiological changes produced little or no disability and consequently major surgery was not required.

The literature relating to pleuropulmonary amoebiasis has been reviewed with particular reference to the pathogenesis, clinical presentation, relevant investigations, diagnosis, management and prognosis. Findings in this study have been compared with those reported in the literature and my conclusions recorded.

The 15 patients with amoebic pericarditis represent the largest personal series documented to date. The clinical, radiological and electrocardiographic findings, the details of management and progress, have been recorded in these patients. Five are presented as cases of presuppurative amoebic pericarditis and 10 as cases of suppurative amoebic pericarditis.

The average duration of symptoms in this group was 10 weeks. A major symptom was low retrosternal and epigastric pain which in some radiated to the left shoulder and was associated with dyspnoea and a non-productive cough. One patient admitted to a past history of dysentery.

Moderate pyrexia, pale mucosae, evidence of weight loss and ankle and sacral oedema were findings seen on general examination. On local examination 11 of the group presented with signs suggestive of a pericardial effusion and evidence of cardiac tamponade. Two patients developed signs of pericarditis following admission for a left lobe liver abscess and in the remaining 2 the diagnosis was missed. Guarding and tenderness in the epigastrium and a tender hepatomegaly were found in each member of the group and in 9 a localised area of maximal tenderness was found in the epigastrium.

Anaemia of a normocytic normochromic variety was a finding in 13 patients. The white cell count was raised in 12 and the sedimentation rate invariably elevated.

Radiologically the cardiothoracic ratio was increased in 12 patients and in 9 there was a globular cardiac outline. In 4 elevation of the left diaphragm was regarded as evidence of associated subphrenic pathology.

Common electrocardiographic changes were generalised T-wave inversion and S-T segment elevation.

Aspiration of pus from a left lobe liver abscess established

/the ...

the source of the pericardial lesion in each of the 5 patients with presuppurative amoebic pericarditis. In 2 of these the presuppurative nature of the pericardial effusion was proved by the aspiration of serosanguineous fluid from the pericardium. In the remaining 3 the diagnosis of presuppurative rather than suppurative pericarditis was based on the clinical presentation and response to treatment.

In 2 of the 10 patients with suppurative amoebic pericarditis the condition was first recognised at necropsy. The suppurative nature of the pericardial effusion was proved in the remaining 8 by the aspiration of pus from the pericardium. In 3 of these 8 patients the diagnosis was suggested by the anchovy sauce character of the pericardial aspirate and in a further 3 it was proved by the identification of E.histolytica in this aspirate. In the remaining 2 aspiration of pus from a left lobe liver abscess and the pericardium suggested the diagnosis. The source of the pericardial lesion was established in 5 of the 10 patients with suppurative amoebic pericarditis by the aspiration of pus from a left lobe liver abscess.

Emetine and chloroquine formed the basis of therapy in this group and pericardial and liver pus was removed by needle aspiration. Repeated aspirations were required to relieve cardiac tamponade in those with suppurative pericarditis. Surgical drainage of the pericardium was attempted in only one instance.

Each of the 5 patients with presuppurative amoebic pericarditis made a complete recovery. Four of the 10 patients with suppurative

/amoebic ...

amoebic pericarditis died, 2 because the diagnosis was missed and 2 following attempted pericardectomy for constrictive amoebic pericarditis. The remaining 6 with suppurative pericarditis made a complete recovery, one of these following a pericardectomy for constrictive amoebic pericarditis.

The literature relating to amoebic pericarditis has been reviewed with particular reference to the pathogenesis, clinical presentation, relevant investigations, diagnosis, management and prognosis. Findings in this study have been compared with those reported in the literature and my conclusions recorded.

APPENDIX A

CASE REPORTS OF 50 PATIENTS WITH PLEUROPULMONARY AMOEBIASIS

Case Report No. 1

Patient M.S. African male Age 59 yrs. Occupation labourer

History: Pleuritic pain in the right lower axilla, a continuous dull pain right upper quadrant and a cough productive of reddish brown sputum for 2 weeks. Past history of dysentery 2 years prior to admission.

Examination: Temperature 99°F. Pale mucosae.
Chest: Sputum, anchovy sauce pus, approximately 200 ml. coughed up on the 1st day. Diminished movement, dullness on percussion, decreased air entry with coarse crepitations at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant, liver edge not palpated.
C.V.S.: An early diastolic murmur at the left sternal border.

Investigations: Hb 9.9 g.%. W.B.C. 6000 per c.mm. E.S.R. 69 mm. in one hour. W.R. -ve. Peripheral smear: Normochromic normocytic anaemia.
Chest Radiographs: Cavity with an air-fluid level at the right base. It was impossible to ascertain whether this cavity was above or below the diaphragm.
Sputum: E.histolytica present. Klebsiella pneumoniae cultured.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage.

Progress: Chest screening showed the abscess cavity to be situated in the right lower lobe, with elevation and immobility of the right hemi-diaphragm. Bronchography showed normal filling of the right lower lobe bronchi with crowding of the branches of the anterior segment. A communication with a subdiaphragmatic abscess was not demonstrated.

The temperature settled on the 3rd day, right upper quadrant tenderness was no longer present on the 5th day, and sputum production ceased in the 2nd week. Serial radiographs showed progressive healing of the abscess cavity but the right hemidiaphragm remained elevated and there were residual patchy changes in the basal segments of the right lower lobe.

The patient recovered completely and was discharged in the 8th week. There had been a weight increase of 10 pounds, the Hb rose to 12 g. per cent and a final W.B.C. was 8000 per c.mm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the diaphragm with formation of a right basal amoebic lung abscess.

Diagnosis: Amoebic liver abscess and amoebic lung abscess.

Case Report No. 2

Patient M.S. African male Age 39 yrs. Occupation labourer

History: Pain in the right lower chest with a cough productive of thick yellowish blood-stained sputum for 3 weeks. Associated symptoms were abdominal pain with diarrhoea blood and mucus for one week. No past history of dysentery.

Examination: Temperature 104°F. Ill-looking. Wasted. Pale mucosae. Finger clubbing.
Chest: Sputum, anchovy sauce pus. Diminished movement, dullness on percussion with decreased air entry at the right base and coarse crepitations at both bases posteriorly.
Abdomen: Generalised abdominal tenderness. The liver was not enlarged. Proctoscopy showed multiple small rectal ulcers with a hyperaemic mucosa.

Investigations: Hb 10 g.%. W.B.C. 12000 per c.mm. E.S.R. 31 mm. in one hour.
Chest Radiographs: Abscess cavity with surrounding consolidation in the apical segment of the right lower lobe. No elevation of the right diaphragm.
Sputum: E.histolytica not detected, heavy growth of Klebsiella pneumoniae cultured.
Stool: Blood, pus and mucus. E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 2nd day and postural drainage. Penicillin for 2 weeks followed by tetracycline for 2 weeks.

Progress: On the 5th day the abdomen became distended with generalised tenderness and ileus developed indicating possible perforation of an amoebic ulcer. Intravenous fluid and gastric suction with intravenous tetracycline were employed until bowel sounds returned on the 10th day.

At the end of the 3rd week pain was experienced in the right upper quadrant and the liver edge palpated 2 fingerbreadths below the right costal margin. From an area of maximum tenderness in the right upper quadrant 360 ml. of typical anchovy sauce pus was aspirated. No amoebae were found in the pus which was sterile on culture for pyogenic organisms. Forty ml. of lipiodol was injected into the abscess cavity but no communication with the right chest could be demonstrated.

Signs in the right upper quadrant were no longer present in the 5th week and the temperature settled in the 9th week. Small quantities of blood-stained sputum were produced daily for 12 weeks.

Bronchoscopy during the 9th week was normal. A bronchogram showed slight fusiform bronchiectasis of the proximal middle lobe bronchi and a collection of contrast media outlined the cavity in the apical segment of the right lower lobe showing it to be decreasing in size.

At discharge during the 12th week crepitations were heard at the right base and small quantities of blood-stained sputum coughed. The patient gained 19 pounds in hospital. The Hb rose to 14 g. per cent and the W.B.C. fell to 5000 per c.mm. Radiographs showed a small persisting cavity with surrounding consolidation in the apical segment of the right lower lobe.

The patient attended follow-up clinic for one year during which time he remained well. A radiograph after one month showed the cavity in the right lower lobe to have healed.

Comments: The peritonitis was considered to have resulted from perforation of an amoebic ulcer. The right lower lobe lung abscess and bronchiectasis were considered to have developed following extension of an amoebic liver abscess through the right diaphragm.

Diagnosis: Amoebic dysentery with peritonitis, amoebic liver abscess, amoebic lung abscess and bronchiectasis.

Case Report No. 3

Patient F.G. African male Age 27 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with radiation to the right shoulder and down the right arm for 6 months. Associated symptoms were cough productive of yellow sputum, weight loss and diarrhoea with blood and mucus. No past history of dysentery.

Examination: Temperature 99°F. Ill-looking. Wasted. Pale mucosae. Chest: Dullness on percussion, decreased air entry with bronchial breathing and crepitations at the right base. Localised intercostal tenderness in the right 9th interspace in the mid-axillary line. Abdomen: Guarding and tenderness right upper quadrant. Liver 3 fingerbreadths enlarged and tender.

Investigations: Hb 3.9 g.%. W.B.C. 20,000 per c.mm. E.S.R. 84 mm. in one hour. Peripheral smear: Normochromic normocytic anaemia. Chest Radiographs: Extensive consolidation in the right middle and lower lobes and patchy consolidation in the left upper lobe. The height of the right diaphragm could not be determined.
Stool: E.histolytica present.

Treatment: Crystalline penicillin was started on admission and packed cells administered. Emetine and chloroquine and diodoquin from the 2nd day.

Progress: Ten ml. of pus in which E.histolytica were identified was aspirated through the right 9th interspace on the 2nd day. The patient's condition deteriorated rapidly and he died on the 3rd day.

Necropsy Findings: A large liver abscess approximately 12 cm. in diameter was found to have eroded through the right diaphragm. This abscess had extended into the right lower lung so that approximately half of the abscess cavity was situated in pulmonary tissue. The upper surface of the liver, the diaphragm and the pleural layers were adherent. The right perinephric tissues were bound to the right lobe of liver by adhesions. There was an abscess of 3 cm. in diameter in the apical region of the left lung surrounded by multiple small abscesses. Examination of the large bowel revealed extensive ulceration.

At histology trophozoites of E.histolytica were identified in the walls of the hepatopulmonary abscess and in the margins of the ulcers in the large bowel. E.histolytica were not found in sections of the abscesses in the upper lobe of the left lung.

Comments: An amoebic liver abscess had eroded through the diaphragm into the base of the right lung. The lung abscesses in the left upper lobe if they were of amoebic origin could have resulted from either

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spread from the right base or haematogenous spread from the liver abscess or bowel.

Diagnosis: Amoebic colitis, hepatopulmonary amoebic abscess with multiple lung abscesses.

Case Report No. 4

Patient G.N. African male Age 44 yrs. Occupation labourer

History: Cough productive of reddish brown blood-stained sputum for 8 months associated with pain in the right shoulder for 6 months. The patient had been admitted on 3 occasions during the past 3 years. On his first admission the diagnosis was amoeboma of ileo-caecal region with amoebic dysentery. On his second admission the diagnosis was amoebic dysentery and on the third occasion signs and radiological changes at the right base were suggestive of bronchiectasis.

Examination: Temperature 101°F. Ill-looking. Clubbing of the fingers.
Chest: Sputum, more than 500 ml. of anchovy sauce pus produced on the first day. Diminished movement, stony dullness and decreased air entry at the right base.
Abdomen: Liver edge not palpable. No tenderness in the right upper quadrant.

Investigations: Hb 13 g.%. W.B.C. 13,000 per c.mm. E.S.R. 44 mm. in one hour. Peripheral Smear: Normochromic normocytic anaemia.
Chest Radiographs: Circumscribed semicircular area of consolidation in the right lower lobe with its lower edge formed by the right diaphragm which was not elevated.
Sputum: E.histolytica present. Sterile on culture for pyogenic bacteria.
Stool: E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 3rd day and postural drainage.

Progress: There was a satisfactory response to treatment. The patient's temperature settled on the 4th day and sputum production ceased at the end of the first week.

A further radiograph on the 12th day showed that the area of consolidation at the right base had broken down with cavity formation.

The patient was discharged during the 3rd week free from symptoms and signs. The Hb on discharge was 13.6 g.% and the W.B.C. 6,000 per c.mm.

He attended after 2 months and was found to be well with no symptoms or signs at the right base. A radiograph showed that the cavity had healed with residual patchy changes in this region.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess.

Case Report No. 5

Patient B.F. African male Age 38 yrs. Occupation labourer

History: There had been cough productive of blood-stained purulent sputum with pleuritic pain in the right lower chest for 4 weeks. There was no past history of dysentery.

Examination: Temperature 99.8°F. Clubbing of the fingers.
Chest: Sputum, blood-stained pus. Diminished movement, dullness on percussion, decreased air entry with coarse crepitations at the right base.
Abdomen: There was no abdominal tenderness and the liver was not enlarged.

Investigations: Hb 10.6 g.%. W.B.C. 19,000 per c.mm. E.S.R. 55 mm. in one hour. Peripheral smear: Normochromic normocytic anaemia.
Chest Radiographs: There was a large abscess cavity with an air-fluid level and surrounding consolidation adjacent to the diaphragm in the right lower lobe. Elevation of the right diaphragm was present.
Sputum: E.histolytica present.
Stool: E.histolytica present.

Treatment: Dehydroemetine, chloroquine, tetracycline from the 3rd day, and postural drainage.

Progress: There was a satisfactory response to treatment. Sputum production ceased on the 6th day and the temperature settled on the 7th day. At this time symptoms were absent but signs persisted at the right base.

Chest radiographs in the 3rd week showed the cavity to be much smaller with resolution of surrounding consolidation and absence of the fluid level. Chest screening showed the right hemidiaphragm to be elevated with sluggish movement and a paradoxical swing on sniffing.

On discharge at the end of the 5th week there were no signs at the right lung base, or in the right upper abdomen, and there had been a twelve pound weight increase. The Hb was 13.4 g.% with a W.B.C. of 7,000 per c.mm. and an E.S.R. of 12 mm. in one hour. Chest radiographs showed the cavity to have healed with residual patchy changes and elevation of the right diaphragm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess.

Case Report No. 6

Patient L.N. African male Age 29 yrs. Occupation labourer

History: Cough productive of thick, yellow sputum for one month and haemoptysis for 2 days. Associated symptoms were generalised malaise, weight loss, anorexia and pain in the left upper quadrant. No past history of dysentery.

Examination: Temperature 99°F. Ill-looking. Pale mucosae. Ankle oedema.
Chest: Sputum, frank blood. Dullness and diminished air entry at the left base.
Abdomen: Slight distension but no tenderness or palpable liver edge.
C.V.S.: Normal. B.P. 120/85 mm. Hg. Examination of fundi showed 2 small haemorrhages with pale centres in the right fundus.

Investigations: Hb 6 g.%. W.B.C. 18,000 per c.mm. E.S.R. 72 mm. in one hour. Peripheral smear: Microcytic hypochromic anaemia.
Chest Radiographs: Minimal patchy consolidation and a pleural reaction at the left base.
Stool: E.histolytica not detected.
Sputum: On direct examination and culture 6 specimens revealed no acid-fast bacilli or pyogenic organisms and examination of 3 specimens no E.histolytica.

Treatment: Penicillin, erythromycin, tetracycline and chloromycetin were the antibiotics used at various stages of the illness in an attempt to control the symptoms. Repeated blood transfusions and oral iron were included in the treatment. In the 6th week emetine, diodoquin and chloroquine were started.

Progress: The patient was intensively investigated for his respiratory symptoms and the anaemia. Bronchoscopy showed blood oozing from the left lower lobe bronchus. It was not until the 6th week that a barium meal abnormality suggested the diagnosis. During this period he remained ill with a high pyrexia and continued to produce large quantities of purulent sputum.

In the 6th week screening of the barium-filled stomach showed a pressure deformity of the fundus and on chest screening the left diaphragm was elevated and fixed with adjacent left basal changes. The above radiological findings suggested a left lobe liver abscess. Aspiration through the left 9th interspace yielded 500 ml. of dark brown pus in which trophozoites of E.histolytica were identified.

A few days following aspiration signs of a large left pleural effusion developed which was confirmed radiologically. Rupture of a left lobe liver abscess into the left pleural cavity was confirmed by thoracentesis. The left chest was aspirated 5 times within the next month and 588 ml. of pus removed which was bacteriologically sterile

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and in which no amoeba was found. Serial radiographs showed that a left pneumo-hydrothorax developed with absorption of the fluid and air after one month.

Following anti-amoebic treatment there seemed to be gradual improvement but haemoptysis, severe anaemia, and signs at the left base persisted. Repeated transfusions were required to maintain the Hb at a level of 6 g. In the 12th week there was evidence of deterioration and the patient died in the 13th week.

Necropsy Findings: The visceral and parietal pleura at the base of the left lung were thickened and adherent. The left diaphragm, the left lobe of the liver and the fundus of the stomach were bound together by dense adhesions.

There was an abscess cavity in the lower lobe of the left lung which communicated with the fundus of the stomach through an opening of approximately 2 cm. in diameter. In the left lobe of the liver immediately underlying the lung abscess was an area of scar tissue approximately 2 cm. in diameter. This was considered to represent the remains of a healed left lobe abscess.

Histology: Trophozoites of E.histolytica were not identified in sections of the scar tissue in the liver or in the lung abscess.

Comments: At necropsy there was evidence that a left lobe amoebic liver abscess had extended through the left diaphragm producing an amoebic empyema and basal lung abscess and that the left basal lung abscess subsequently extended into the stomach with formation of a pulmonary gastric fistula.

Diagnosis: Left lobe amoebic liver abscess, amoebic empyema, amoebic lung abscess with a pulmonary gastric fistula.

Case Report No. 7

Patient T.M. African male Age 27 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with radiation to the right shoulder for 4 months. Associated symptoms were cough productive of blood-stained yellow sputum in large quantities and a dull continuous pain in the right upper quadrant. Chest radiographs 4 months prior to admission showed consolidation of the right lower lobe and there was no improvement following treatment with antibiotics. No past history of amoebic dysentery.

Examination: Temperature subnormal. Pale mucosae. Clubbing of the fingers and toes.
Chest: Sputum, anchovy sauce pus approximately 300 ml. on the first day. Diminished movement, stony dullness on percussion and decreased air entry at the right base. Bronchial breathing heard in the right lower axilla.
Abdomen: Tenderness and guarding in the right upper quadrant and the liver edge palpated 2 fingerbreadths below the right costal margin.

Investigations: Hb 8.7 g.%. W.B.C. 12,000 per c.mm. E.S.R. 57 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Slight elevation of the right diaphragm. Large cavity with surrounding consolidation and an air-fluid level in the posterior aspect of the right lower lobe in close proximity to the diaphragm.
Sputum: E.histolytica not detected. Normal respiratory flora cultured from 6 specimens.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin and postural drainage from the 2nd day. A course of penicillin was added to the above regime.

Progress: The patient remained apyrexial throughout hospitalisation. For the first 2 weeks he continued to cough decreasing quantities of typical anchovy sauce sputum. The bronchial breathing at the right base disappeared but dullness and decreased air entry persisted. The liver edge was no longer palpable and right upper quadrant signs cleared within one week.

Serial radiographs showed a progressive decrease in the size of the cavity with clearing of surrounding consolidation.

At discharge on the 27th day there was slight dullness on percussion at the right base with no symptoms. The Hb had risen to 15.2 g.%, the W.B.C. was 10,000 per c.mm. and the E.S.R. 22 mm. in one hour. There had been a weight increase of 8 pounds. Chest radiographs showed a small remaining cavity at the right base and no residual elevation of the right diaphragm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and amoebic lung abscess.

Case Report No. 8

Patient Z.N. African male Age 34 yrs. Occupation labourer

History: Cough productive of blood-stained sputum for 3 months associated with a pleuritic pain in the right lower chest referred to the right shoulder. No past history of dysentery.

Examination: Temperature 100°F. Pale mucosae. Finger clubbing.
Chest: Sputum, frank blood and blood-stained pus. Dullness on percussion with diminished air entry at the right base. Vocal resonance increased and coarse crepitations at the right base.
Abdomen: No tenderness in the right upper quadrant. Liver not enlarged.

Investigations: Hb 8.6 g.%. W.B.C. 28,000 per c.mm. E.S.R. 61 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Opacity at the right base which on the lateral film appeared to be due to marked localised elevation of the posterior aspect of the right diaphragm. Fluid levels were seen in this opacity and it could not be determined whether they were above or below the diaphragm.
Sputum: E.histolytica present. Sterile on culture for pyogenic bacteria.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin and postural drainage from the 2nd day.

Progress: The temperature settled on the 5th day and sputum production ceased after one week's treatment. Crepitations cleared at the right base but dullness on percussion and decreased air entry persisted until discharge.

The E.S.R. and W.B.C. fell to 26 mm. in one hour and 8,000 per c.mm. and the Hb rose to 11 g.%.

Serial radiographs showed that the opacity at the right base with the air-fluid level was above the right diaphragm.

At discharge the prominent posterior elevation of the right diaphragm was no longer present but there was moderate general residual elevation. An abscess cavity was no longer visible and there were residual patchy changes in the right lower lobe adjacent to the diaphragm.

The patient was discharged on the 26th day free of symptoms with dullness on percussion at the right base. The weight had increased by 8 pounds. At follow-up clinic after one month clinical progress was maintained. Chest radiographs showed the right diaphragm to be elevated with minimal adjacent patchy changes.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and amoebic lung abscess.

Case Report No. 9

Patient R.S. African male Age 27 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest for 2 days. On the day prior to admission quantities of reddish-brown sputum were coughed. No past history of dysentery.

Examination: Temperature 102°F. Ill-looking. Pale mucosae.
Chest: Soon after admission suddenly coughed up approximately 400 ml. of anchovy sauce pus. Dullness on percussion, diminished air entry and coarse crepitations at the right base.
Abdomen: Tenderness in the right upper quadrant with a 2 fingerbreadths tender, enlarged liver.

Investigations: Hb 8.3 g.%. W.B.C. 17,000 per c.mm. E.S.R. 62 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest radiographs: Consolidation of the lower half of the right lower lobe. The right diaphragm was in normal position with a small interlobar effusion at the base of the greater fissure.
Sputum: E.histolytica present. Moderate growth of B.coli reported on culture.
Stool: E.histolytica absent.

Treatment: Emetine, chloroquine and diedoquin with postural drainage from the 3rd day. A 10-day course of penicillin was included in the treatment.

Progress: Decreasing quantities of anchovy sauce sputum were produced until the 6th day when sputum production ceased. The temperature settled on the 10th day. A pleural rub was heard at the right base on the 3rd day. Liver enlargement and tenderness resolved within one week.

At discharge on the 20th day there were no residual local signs. The patient gained 9 pounds in hospital, the Hb rose to 10.8 g.%, the W.B.C. fell to 10,000 per c.mm. and the E.S.R. to 18 mm. in one hour.

Chest radiographs at discharge showed residual patchy consolidation in the right lower lobe with a small cavity, the interlobar effusion had cleared and shadowing adjacent to the diaphragm suggested a pleural reaction.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess.

Case Report No. 10

Patient M.S. African male Age 40 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with radiation to the right shoulder for 3 weeks. Associated complaints were cough, tiredness, anorexia and nausea. There was no past history of dysentery.

Examination: Temperature 103°F. Ill-looking. Pale mucosae.
Chest: Diminished movement, dullness on percussion, decreased air entry and localised intercostal tenderness at the right base.
Abdomen: Slight abdominal distension with tenderness and guarding in the right upper quadrant. Liver 4 fingerbreadths enlarged.

Investigations: Hb 10.9 g.%. W.B.C. 17,000 per c.mm. E.S.R. 69 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Marked elevation of the right diaphragm with irregularity of contour and a pleural reaction at the right base.
Liver Aspirate: E.histolytica present. Bacteriologically sterile.
Sputum: E.histolytica not detected. Sterile on culture for pyogenic bacteria.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin and postural drainage from the 1st day.

Progress: Progress was slow during the 1st week. Three liver aspirations were done through the 9th interspace in the mid-axillary line and a total of 150 ml. of thick, yellow pus aspirated without relieving symptoms.

On the 11th day the patient coughed up large quantities of thick, anchovy sauce sputum and developed signs of consolidation at the right base. Chest radiographs at this stage showed the right diaphragm to be returning to normal levels and a cavity with surrounding consolidation in the posterior inferior aspect of the right lower lobe. Bronchoscopy showed no abnormality of right middle and lower lobe orifices.

Sputum production ceased on the 15th day and the temperature settled on the 20th day. The patient was discharged during the 6th week. The liver was no longer palpable but there was persisting dullness and diminished air entry at the right base. The Hb rose to 11.9 g.%, the W.B.C. and E.S.R. fell to 10,000 per c.mm. and 18 mm. in one hour respectively. Chest X-ray showed that the cavity had healed with residual patchy changes, the diaphragm remained slightly elevated and minimal adjacent pleura thickening was thought to be present.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess.

Case Report No. 11

Patient G.B. African male Age 48 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with fever for one week. The day before admission a large quantity of blood-stained sputum was coughed. No past history of dysentery.

Examination: Temperature 99°F. Pale mucosae.
Chest: Sputum, frank blood and blood-stained pus. Dullness on percussion, decreased air entry with crepitations posteriorly and a pleural rub at the right base. Localised intercostal tenderness in this region.
Abdomen: Tenderness and guarding in the right upper quadrant, liver edge not palpable.

Investigations: Hb 10.6 g.%. W.B.C. 18,000 per c.mm. E.S.R. 57 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: The right hemidiaphragm was not elevated and there was a small circumscribed semicircular area of consolidation adjacent to the diaphragm in the posterior aspect of the right lower lobe.
Liver Aspirate: E.histolytica not detected. Sterile on culture.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Crystalline penicillin 1 million units q.i.d. was commenced with no response after 5 days. Emetine, chloroquine and diodoquin were started on the 5th day and postural drainage.

Progress: There was no response to penicillin but the temperature settled 24 hours following treatment with emetine and chloroquine. Liver aspiration on the 4th day yielded 5 ml. of thick, blood-stained pus. Symptoms and signs in the right upper quadrant with sputum production cleared by the 9th day but signs persisted at the right base. Chest radiographs at this time showed that the area of consolidation at the right base had cavitated.

The patient was discharged on the 20th day. The Hb at discharge was 10.7 g.% and the W.B.C. 12,000 per c.mm.

One month later he remained well. There were no signs in the chest and a radiograph showed that the cavity had healed.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess and amoebic lung abscess.

Case Report No. 12

Patient T.N. African male Age 25 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with a cough productive of brownish sputum for 4 weeks. Four months prior to admission an episode of dysentery which lasted 2 months.

Examination: Temperature 102°F. Ill-looking. Pale mucosae.
Chest: Sputum, approximately 300 ml. of anchovy sauce pus on the first day. Diminished movement, dullness on percussion, decreased air entry with localised intercostal tenderness at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant, with a tender liver edge palpated 2 fingerbreadths below the right costal margin.

Investigations: Hb 6.5 g.%. W.B.C. 20,000 per c.mm. E.S.R. 61 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Elevation of the right hemidiaphragm with a small adjacent area of circumscribed semicircular consolidation in the right lower lobe.
Sputum: E.histolytica not detected. Klebsiella pneumoniae cultured.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage. Ten-day courses of penicillin and tetracycline were added to the above regime.

Progress: The temperature settled on the 7th day and from one to two hundred ml. of blood-stained sputum was produced daily for 2 weeks.

Liver aspiration was attempted unsuccessfully during the 2nd week. Chest radiographs at this time showed an air-fluid level below the right diaphragm with an abscess cavity in the right lower lobe and a small encysted effusion at the base of the greater fissure. A bronchogram showed normal filling of the bronchial segments of the right middle and lower lobes, and no abnormality was found at bronchoscopy.

Tachycardia developed in the 4th week in the absence of symptoms or pyrexia. Emetine toxicity was considered. An E.C.G. showed no abnormality and the pulse rate settled in the 7th week.

At discharge during the 8th week there had been a weight increase of 14 pounds, dullness with diminished air entry persisted at the right base and liver enlargement and tenderness were not detected. The Hb was 13.7 g.%, the W.B.C. 8,000 per c.mm. and the E.S.R. 6 mm. in one hour. Chest radiographs showed that the abscess cavities in the liver and right lower lobe had healed with clearing of the encysted effusion. There was residual elevation of the diaphragm with minimal adjacent shadowing suggestive of a pleural reaction.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the base of the right lung with formation of an amoebic lung abscess. The air-fluid level below the right diaphragm proved the existence of an hepatobronchial fistula.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess with an hepatobronchial fistula and an encysted effusion.

Case Report No. 13

Patient E.M. African male Age 40 yrs. Occupation labourer

History: Stabbing pain in the right lower chest with radiation to the right shoulder for 2 weeks. Associated symptoms were cough, dyspnoea, pain in the right upper quadrant and profuse sweating. No past history of dysentery.

Examination: Temperature 102°F. Ill-looking.
Chest: Diminished movement, dullness on percussion, decreased air entry with localised intercostal tenderness at the right base.
Abdomen: Guarding and tenderness in the right upper quadrant with a tender liver edge palpated 2 fingerbreadths below the costal margin.

Investigations: Hb 10 g.%. W.B.C. 21,000 per c.mm. E.S.R. 57 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Marked elevation of the right hemidiaphragm with minimal adjacent patchy consolidation and a small effusion in the right costophrenic angle.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine, diodoquin. From the 2nd day, a 10-day course of penicillin and postural drainage.

Progress: On the 4th day approximately 700 ml. of anchovy sauce pus was suddenly coughed up. From 50 to 200 ml. of sputum was coughed daily until the end of the 3rd week. The temperature settled on the 9th day and by this time there was absence of pain and tenderness with the liver no longer palpable.

During hospitalisation signs of a right sided effusion developed and serial radiographs showed this to be loculated and situated in the right upper chest. Penetrated radiographs showed a cavity with an air-fluid level below the loculated effusion in the apical segment of the right lower lobe. Thoracentesis yielded 20 ml. of serosanguineous fluid. A bronchogram showed no hepatobronchial fistula.

At discharge during the 7th week signs persisted at the right base. There was a weight increase of 11 pounds. Chest radiographs showed moderate residual elevation of the right diaphragm with a noticeable decrease in the loculated effusion, resolution of the cavity in the apical segment and shadows suggestive of pleural thickening at the right base. The Hb had risen to 12.8 g.% and the W.B.C. and E.S.R. fell to 7,000 per c.mm. and 22 mm. in one hour, respectively.

Comments: The clinical picture was interpreted as being that of an

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amoebic liver abscess which had extended through the right diaphragm with formation of an amoebic lung abscess. The pleural effusion possibly resulted from the inflammatory reaction at the periphery of the lung abscess.

Diagnosis: Amoebic liver abscess, amoebic lung abscess with a pleural effusion.

Case Report No. 14

Patient S.K. African male Age 29 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest for 2 weeks with radiation to the right shoulder. Cough productive of large amounts of blood-stained sputum. Treated for amoebic dysentery 3 months prior to admission.

Examination: Temperature 100°F. Pale mucosae.
Chest: Sputum, quantities of blood-stained dirty yellow non-offensive pus. Dullness, diminished air entry with localised intercostal tenderness at the right base.
Abdomen: Guarding and tenderness in the right upper quadrant and a tender liver edge palpated 4 fingerbreadths below the costal margin.

Investigations: Hb 8.7 g.%. W.B.C. 12,000 per c.mm. E.S.R. 61 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Elevation of the right hemidiaphragm with a small pleural effusion at the right base. A cavity with an air-fluid level was present in the basal portion of the right lower lobe.
Sputum: E.histolytica present.
Stool: E.histolytica not detected.

Treatment: Dehydroemetine, chloroquine and diodoquin from the first day and postural drainage.

Progress: Trophozoites of E.histolytica were seen on direct examination of the sputum on the 2nd day. Fifty ml. of straw-coloured fluid was aspirated through the 9th interspace in the posterior axillary line on the 5th day. There were no cells or organisms detected in the aspirate which was sterile on culture for bacteria and acid-fast bacilli and had a protein content of 3.9 g.%.

The temperature settled on the 3rd day and sputum production ceased on the 9th day by which time tenderness was no longer present in the right upper quadrant and the liver edge no longer palpable.

At discharge on the 24th day dullness and diminished air entry persisted at the right base. There was a weight increase of 4 pounds and the Hb had risen to 10.3 g.% and the W.B.C. and E.S.R. were 8,000 per c.mm. and 29 mm. in one hour respectively. Chest radiographs showed clearing of the right basal changes with the right diaphragm in normal position.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm with formation of an amoebic lung abscess.

Diagnosis: Amoebic liver abscess, and an amoebic lung abscess with a pleural effusion.

Case Report No. 15

Patient E.Z. African male Age 30 yrs. Occupation labourer

History: Ill for one month with pain in the right lower chest radiating to the right neck. No past history of dysentery.

Examination: Apyrexial.
Chest: Diminished movement, percussion note, and air entry at the right base with localised intercostal tenderness.
Abdomen: No tenderness in the right upper quadrant. Liver 2 finger-breadths enlarged, non tender, with a firm edge.

Investigations: Hb 12.5 g.%. W.B.C. 25,000 per c.mm. E.S.R. 33 mm. in one hour.
Chest Radiographs: Elevation of the right hemidiaphragm with an adjacent area of circumscribed semicircular consolidation.
Sputum: Trophozoites of E.histolytica present.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 4th day and postural drainage.

Progress: On the 3rd day the patient suddenly coughed approximately 250 ml. of anchovy sauce pus in which trophozoites of E.histolytica were detected. The signs at the right base increased and the temperature rose to 103°F. Chest radiographs at this stage showed the right diaphragm to be in normal position with extensive consolidation of the right lower lobe.

There was no further sputum production after the 4th day, the temperature settled on the 8th day and symptoms cleared by the 12th day.

At discharge during the 5th week there had been a weight increase of 4 pounds, dullness and decreased air entry persisted at the right base, and there was no change in the size of the liver. The Hb rose to 13.8 g.% and the W.B.C. fell to 10,000 per c.mm. Chest radiographs at this stage showed the diaphragm to be in normal position with a small adjacent area of residual consolidation. The patient failed to attend follow up clinic.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 16

Patient A.L. African male Age 22 yrs. Occupation labourer

History: Four months prior to admission experienced an episode of diarrhoea which lasted 3 weeks. This was followed by onset of pleuritic pain which involved the right lower chest and epigastrium. Cough with haemoptysis developed one month before admission.

Examination: Temperature 100°F. Ill-looking. Evidence of recent weight loss.
Chest: Sputum, blood-stained pus. Diminished movement, dullness on percussion and decreased air entry with localised intercostal tenderness at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant and a tender liver edge palpated 2 fingerbreadths below the costal margin.

Investigations: Hb 11.8 g.%. W.B.C. 6,000 per c.mm. E.S.R. 54 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: The diaphragm was in normal position, the lung fields clear with no evidence of a pleural reaction at the right base.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage.

Progress: The temperature settled on the 3rd day and sputum production progressively diminished ceasing on the 9th day. The patient was discharged at the end of the 2nd week free of symptoms and signs. The Hb at discharge was 13 g.% and the W.B.C. 7,000 per c.mm. Chest radiographs remained normal.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus. There were no radiological changes associated with this event.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 17

Patient K.M. African male Age 19 yrs. Occupation labourer

History: Pain of a pleuritic nature in the right lower chest associated with a cough productive of large quantities of blood-stained yellow sputum for 3 days. No past history of dysentery.

Examination: Apyrexial.
Chest: Sputum, blood-stained pus. Diminished movement, air entry and percussion note with a pleural friction rub at the right base.
Localised intercostal tenderness at the right base.
Abdomen: No tenderness. Liver not enlarged.

Investigations: Hb 15 g.%. W.B.C. 5,000 per c.mm. E.S.R. 20 mm. in one hour.
Chest Radiographs: Elevation of the right diaphragm with a small area of circumscribed semicircular consolidation adjacent to the diaphragm in the right lower lobe.
Sputum: E.histolytica present.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 2nd day and postural drainage.

Progress: Liver aspiration was attempted and failed. The patient was apyrexial whilst in hospital, symptoms cleared and sputum production ceased on the 4th day. On discharge during the 2nd week signs suggestive of diaphragmatic elevation were present.

The patient continued to attend hospital for one year and nine months after discharge. He had remained well and the signs at the right base cleared. Chest radiographs showed the diaphragm to be in normal position with resolution of the right basal consolidation.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 18

Patient J.M. African male Age 38 yrs. Occupation labourer

History: Cough productive of reddish-brown sputum with pain in the right shoulder for 5 weeks. There was no pain in the right chest. Past history of having received treatment for an amoebic liver abscess and amoebic dysentery 17 years prior to admission.

Examination: Temperature 99°F. Ill-looking. Pale mucosae.
Chest: Sputum, approximately 600 ml. of anchovy sauce pus coughed up on the first day. Diminished movement, dullness on percussion and absent air entry at the right base.
Abdomen: No tenderness in the right upper quadrant and the liver edge not palpable.

Investigations: Hb 8.4 g.%. W.B.C. 5,000 per c.mm. E.S.R. 64 mm. in one hour. Peripheral smear: Normochromic normocytic anaemia.
Chest Radiographs: Complete opacification of the right middle lobe area with bulging of the lesser and greater fissures. Patchy consolidation of the right lower lobe with elevation of the right hemidiaphragm.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 4th day and postural drainage. A 10-day course of penicillin was added.

Progress: Anchovy sauce sputum was coughed up in decreasing quantities for the first 12 days, at which stage sputum production ceased and the temperature settled.

The signs at the right base gradually became less noticeable until at discharge on the 20th day only slight dullness remained. There was a weight increase of 5 pounds during hospitalisation.

The Hb rose to 10.2 g.% and the W.B.C. remained at 5,000 per c.mm. Serial radiographs showed partial clearing of the middle lobe consolidation, complete clearing of the consolidation in the right lower lobe and persistent elevation of the right diaphragm.

At follow-up clinic after one month the patient remained well and symptom free. There was still detectable dullness on percussion at the right base. Chest radiographs showed residual elevation of the right diaphragm with further clearing of the middle lobe consolidation. A right lateral view indicated that partial collapse of the middle lobe persisted.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus. The radiological changes were probably the consequence of inflammatory reaction surrounding an hepatobronchial fistula.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 19

Patient S.K. African male Age 31 yrs. Occupation labourer

History: Pain in the right lower chest and right upper quadrant for 4 weeks. Two days before admission the pain worsened and blood-stained brownish sputum was coughed. There was no past history of dysentery.

Examination: Apyrexial. Pale mucosae.
Chest: Sputum, large quantities of blood-streaked anchovy sauce pus. Dullness on percussion, diminished air entry and crepitations with localised intercostal tenderness at the right base.
Abdomen: Tenderness in the right upper quadrant and a tender liver edge palpated.

Investigations: Hb 10.4 g.%. W.B.C. 8,000 per c.mm. E.S.R. 58 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: The right lower lung field was opaque. This was considered to represent consolidation of the right lower lobe. The level of the right diaphragm was not visualised.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage. Oxytetracycline 250 mg. q.i.d. for 10 days added to above regime.

Progress: Aspiration of the right lower chest was attempted and failed. The patient remained apyrexial throughout his stay and no further sputum was produced after the 4th day. The symptoms and signs in the right upper abdomen cleared within the first week. Bronchoscopy showed the orifices of the right lower and middle lobes to be normal.

At discharge during the 3rd week dullness on percussion and diminished air entry persisted at the right base. The Hb had risen to 12.6 g.% and the W.B.C. was 7,000 per c.mm. with an E.S.R. of 32 mm. in one hour. Chest radiographs showed clearing of the consolidation of the right lower lung field with elevation and irregularity of the right diaphragm persisting. There was minimal residual shadowing in the lung field adjacent to the diaphragm thought to represent pleural thickening.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 20

Patient P.K. African male Age 48 yrs. Occupation labourer

History: Stabbing pain in the right lower chest referred to the right shoulder, for one year. Associated symptoms were cough with blood-stained sputum, profuse sweats and weight loss. There was no past history of dysentery.

Examination: Apyrexial.
Chest: Sputum, blood-stained pus. Diminished percussion note, crepitations and rhonchi with decreased air entry and intercostal tenderness were found at the right base.
Abdomen: Tenderness in the right upper quadrant with the liver edge 2 fingerbreadths enlarged and tender.

Investigations: Hb 15 g.%. W.B.C. 9,000 per c.mm. E.S.R. 20 mm. in one hour.
Chest Radiographs: Slight elevation of the right hemidiaphragm with a small area of circumscribed semicircular consolidation in the adjacent portion of the right lower lobe.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Crystalline penicillin q.i.d. from 4th day. Emetine, chloroquine and diodoquin from 7th day and postural drainage.

Progress: Liver aspiration was attempted without success on the 2nd day. Four days of crystalline penicillin had no effect on the temperature which fluctuated between 99 and 101°F. Twenty-four hours following treatment with emetine and chloroquine the temperature settled and pain and tenderness cleared. Sputum production ceased following one week's treatment and the liver edge was no longer palpable at this stage.

At discharge on the 20th day there were no residual signs at the right base. Radiographs showed the diaphragm to be in normal position with resolution of the adjacent area of consolidation.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 21

Patient M.M. African male Age 28 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest referred to the right shoulder, with a cough productive of small quantities of blood-stained sputum for 5 months. Breathlessness on exertion and diarrhoea during the above period. No past history of dysentery.

Examination: Temperature 101°F. Ill-looking. Wasted. Pale mucosae.
Chest: Sputum, large quantities blood-stained dirty yellow pus. Trachea deviated to the left. Diminished movement, stony dullness on percussion and absent air entry over right lower lung field. Localised intercostal tenderness present.
Abdomen: Tenderness and guarding in the right upper quadrant and a tender liver palpated 2 fingerbreadths below the costal margin.

Investigations: Hb 6.9 g.%. W.B.C. 8,000 per c.mm. E.S.R. 70 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Right hemidiaphragm much elevated with irregular contour, a pleural reaction posteriorly at the right base and patchy changes in the adjacent right lower lung field.
Sputum: E.histolytica not detected.
Stool: E.histolytica present.
Liver Aspirate: E.histolytica not detected. Sterile on culture.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage. A seven-day course of tetracycline was included in the treatment.

Progress: Intercostal liver aspiration on the 2nd day yielded 480 ml. of anchovy sauce pus. At this time 300 ml. of typical anchovy sauce pus was suddenly coughed up.

The temperature settled on the 9th day and liver enlargement and tenderness were absent on the 10th day of treatment.

Radiographs on the 12th day showed that an air-fluid level had developed below the right diaphragm which remained raised. Overlying linear shadowing suggested atelectasis of the adjacent segments of the right lower lobe.

A bronchogram on the 22nd day showed that diaphragmatic elevation with the underlying air-fluid level persisted and that there was upward displacement with crowding of the right lower lobe bronchi. A communication between the liver abscess and the lung was not demonstrated.

The patient was discharged during the 5th week, free of symptoms, having gained 4 pounds. The Hb was 12 g.%, W.B.C. 6,000 per c.mm. and E.S.R. 24 mm. in one hour. Signs of dullness and diminished air entry persisted at the right base. Radiographs of the chest at discharge showed resolution of the air-fluid level below the diaphragm with residual elevation of the diaphragm and adjacent linear shadowing in the right lower lobe.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 22

Patient J.M. African male Age 32 yrs. Occupation labourer

History: Stabbing pain in the right lower chest and right upper quadrant with radiation to the right shoulder for 4 days. No past history of dysentery.

Examination: Temperature 102°F. Ill-looking.
Chest: Diminished movement, dullness, decreased air entry with localised intercostal tenderness at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant. The liver edge was not palpable.

Investigations: Hb 12.4 g.%. W.B.C. 12,000 per c.mm. E.S.R. 28 mm. in one hour.
Chest Radiographs: Elevation of the right diaphragm with a posteriorly situated encysted effusion at the right base.
Liver Aspirate: E.histolytica not detected. Sterile on culture for pyogenic organisms.
Pleural Aspirate: E.histolytica not detected. Sterile on culture for pyogenic organisms and acid-fast bacilli.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage.

Progress: Approximately 500 ml. of blood-stained pus was suddenly coughed up during the third night. On the 4th day 95 ml. of thick yellow pus was aspirated from the liver via the intercostal route.

Radiographs on the 10th day showed the encysted effusion to be still present. This was aspirated and 50 ml. of a thin blood-stained fluid was obtained.

The temperature settled on the 8th day and pain and tenderness with sputum production ceased by the 10th day.

At discharge during the 4th week dullness and diminished air entry persisted at the right base and his weight had increased by 3 pounds. Radiographs showed the diaphragm to be still raised with partial clearing of the effusion. The Hb was 11.4 g.%, the W.B.C. 12,000 per c.mm. and the E.S.R. 14 mm. in one hour.

At follow-up 3 months after discharge the patient was well, there were no signs at the right base. A radiograph showed slight residual posterior elevation of the right diaphragm with shadows suggestive of pleural thickening.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula and a pleural effusion.

Case Report No. 23

Patient M.D. African male Age 36 yrs. Occupation labourer

History: Pain in the right lower chest for one month. Cough productive of blood-stained sputum for 3 weeks. Additional complaints were excessive night sweats and fever. One month prior to admission diagnosed and treated as a right lower lobe pneumonia which failed to resolve clinically and radiologically. No past history of dysentery.

Examination: Temperature 101°F. Pale mucosae. Finger clubbing.
Chest: Sputum, large quantities of anchovy sauce pus. Diminished movement, dullness, decreased air entry, crepitations and localised intercostal tenderness at the right base.
Abdomen: No right upper quadrant tenderness and the liver edge not palpable.

Investigations: Hb 7.1 g.%. W.B.C. 12,000 per c.mm. E.S.R. 67 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Lobar consolidation of the right middle lobe with elevation of the right hemidiaphragm.
Sputum: E.histolytica present on 2 occasions. Haemolytic Streptococci grown on culture.
Stool: E.histolytica not detected.

Treatment: Dehydroemetine, chlorequine and diodoquin from the 2nd day and postural drainage.

Progress: The temperature settled on the 4th day and sputum production ceased on the 11th day. Signs persisted at the right base and bronchial breathing was heard.

Serial radiographs showed increasing resolution of the right middle lobe consolidation with atelectasis. A bronchogram at one month revealed marked atelectasis with early fusiform bronchiectasis of the right middle lobe.

At discharge during the 5th week signs persisted at the right base. The Hb rose to 13.5 g.%, the W.B.C. was 8,000 per c.mm. and the E.S.R. 28 mm. in one hour. Radiographs showed residual elevation of the right diaphragm with a small adjacent area of consolidation which on the lateral view was seen to be due to middle lobe collapse. Permission for bronchoscopy and surgery was refused. There was failure to attend follow-up clinic.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus. The middle lobe atelectasis and bronchiectasis were probably the consequence of the inflammatory reaction in the region of the hepatobronchial fistula.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula and residual middle lobe bronchiectasis.

Case Report No. 24

Patient M.C. African male Age 26 yrs. Occupation labourer

History: Pain of a stabbing nature in the right lower chest with pain in the right upper quadrant for 5 weeks. There was a non-productive cough. No past history of dysentery.

Examination: Temperature 99°F.
Chest: No signs at the right base apart from localised intercostal tenderness in the right 10th interspace at the mid-axillary line.
Abdomen: Tenderness and guarding in the right upper quadrant. Liver edge not palpable.

Investigations: Hb 12.3 g.%. W.B.C. 12,000 per c.mm. E.S.R. 34 mm. in one hour.
Chest Radiographs: Elevation with irregularity of contour of the right hemidiaphragm and a small adjacent area of semicircular consolidation in the right lower lobe.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.
Liver Aspirate: E.histolytica not detected. Sterile on culture for pyogenic organisms.

Treatment: Emetine, chloroquine and diodoquin from the 3rd day and postural drainage.

Progress: On the 2nd day 40 ml. of anchovy sauce pus was aspirated through the area of maximal intercostal tenderness. On the 5th day approximately 900 ml. of anchovy sauce pus was coughed up. Sputum production ceased with settling of the temperature on the 10th day.
At discharge at the end of the 2nd week there were no symptoms or signs. Radiographs showed elevation of the right hemidiaphragm with a minimal adjacent area of consolidation.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 25

Patient M.V. African male Age 22 yrs. Occupation labourer

History: Cough with blood-stained sputum and pleuritic pain in the right lower chest for one week. Pain in the right shoulder and dyspnoea 2 days before admission. No past history of dysentery.

Examination: Temperature 99°F. Ill-looking.
Chest: Sputum, more than 500 ml. of anchovy sauce pus coughed on the 1st day. Movement, percussion note and air entry diminished at the right base with crepitations audible in the right axilla. Localised intercostal tenderness.
Abdomen: Tenderness and guarding in the right upper quadrant, liver edge not palpable.

Investigations: Hb 13.4 g.%. W.B.C. 11,000 per c.mm.
Chest Radiographs: Irregularity in contour of the right hemidiaphragm with anterior elevation, patchy consolidation in the adjacent right lower lobe and lobar consolidation of the right middle lobe.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chlorequine and diodoquin from the 1st day and postural drainage.

Progress: Liver aspiration was attempted and failed. The patient was free from pain on the 4th day and the temperature settled on the 3rd day. Quantities of anchovy sauce sputum were produced in decreasing amounts during the 1st week.

He was discharged at the end of the 2nd week with signs suggestive of diaphragmatic elevation still present. The Hb was 13.6 g.% and the W.B.C. 8,000 per c.mm. Radiographs on discharge showed clearing of the lung changes with a small area of residual patchy shadowing adjacent to the diaphragm and persisting elevation of the anterior aspect of the right diaphragm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus. The radiological pulmonary changes were probably the consequence of the inflammatory reaction in the vicinity of the hepatobronchial fistula.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 26

Patient J.N. African male Age 56 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest, with a cough productive of large quantities of blood-stained sputum for 2 weeks. Associated symptoms were fever and anorexia. No past history of dysentery.

Examination: Temperature 101°F. Ill-looking. Pale mucosae.
Chest: No sputum coughed on admission. Diminished movement, dullness on percussion with decreased air entry and bronchial breathing at the right base. Localised intercostal tenderness in the lower right rib spaces.
Abdomen: Liver not enlarged.

Investigations: Hb 7 g.%. W.B.C. 11,000 per c.mm. E.S.R. 69 mm. in one hour. Peripheral smear: Normocytic, normochromic anaemia.
Chest Radiographs: Elevation of the right hemidiaphragm with adjacent circumscribed consolidation in the basal segments of the right lower lobe and a small interlobar effusion at the right base.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day.

Progress: On admission 60 ml. of anchovy sauce pus was aspirated through the 9th interspace in the anterior axillary line. Three further attempts at aspiration in the first 12 days were unsuccessful.

No sputum was coughed up in hospital and the temperature settled on the 6th day. Intercostal tenderness and bronchial breathing resolved and at no stage were signs found in the right upper quadrant.

Serial chest radiographs showed resolution of the consolidation at the right base and clearing of the effusion, with persisting elevation of the right hemidiaphragm.

At discharge on the 22nd day there had been a weight increase of 8 pounds but dullness and diminished air entry persisted at the right base. The Hb had risen to 10.3 g.%, the W.B.C. was 8,000 per c.mm. and the E.S.R. 56 mm. in one hour.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula and an interlobar effusion.

Case Report No. 27

Patient N.W. African male Age 32 yrs. Occupation labourer

History: Pain in the right upper quadrant, referred to the right shoulder for one month. Cough productive of blood-stained sputum for one week. Three months prior to admission was treated for amoebic dysentery.

Examination: Temperature 100°F.
Chest: Sputum, small quantities of blood-stained pus. Diminished movement, percussion note and air entry with crepitations and localised intercostal tenderness at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant with a 2 fingerbreadths enlarged tender liver.

Investigations: Hb 14.4 g.%. W.B.C. 15,000 per c.mm.
Chest Radiographs: Right hemidiaphragm moderately raised with irregularity of contour. Patchy changes in the right lower lobe adjacent to the diaphragm.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 3rd day and postural drainage.

Progress: The temperature settled on the 3rd day. Several hundred millilitres of anchovy sauce sputum was suddenly coughed up during the 7th night. Symptoms settled with no further sputum production on the 10th day.

At discharge during the 3rd week the patient had gained 8 pounds, signs of right diaphragmatic elevation persisted and right upper abdominal signs had resolved. The Hb was 15 g.% and the W.B.C. 8,000 per c.mm. Radiographs at discharge showed residual elevation of the right diaphragm with clearing of the patchy changes in the adjacent lung field and evidence of a slight pleural reaction at the right base.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 28

Patient S.K. African male Age 23 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with intermittent coughing of blood-stained sputum for 3 months. No past history of dysentery.

Examination: Apyrexial. Ill-looking. Pale mucosae. Finger clubbing. Chest: Sputum, more than 500 ml. of blood-stained pus coughed up on admission. Diminished movement, dullness on percussion with decreased air entry at the right base and marked localised intercostal tenderness in the 9th interspace at the anterior axillary line.
Abdomen: Tenderness and guarding in the right upper quadrant and the liver edge not palpable.

Investigations: Hb 10.1 g.%. W.B.C. 13,000 per c.mm. E.S.R. 57 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Right hemidiaphragm much elevated with linear atelectasis and patchy consolidation at the right base.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and postural drainage. A course of penicillin was added.

Progress: Liver aspiration was attempted and failed. An intermittent pyrexia settled on the 15th day and during this period the patient continued to cough approximately 500 ml. of blood-stained sputum per day. Abdominal signs resolved in the second week. Sputum production ceased in the third week.

Serial radiographs of the chest showed clearing of the pneumonic changes and that an air-fluid level had developed beneath the raised right diaphragm, confirming the presence of a subdiaphragmatic abscess.

There was a slow but satisfactory response to treatment. The patient unfortunately discharged himself prematurely on the 24th day insisting that he had completely recovered. At this stage dullness and diminished air entry persisted at the right base and there had been a weight increase of 8 pounds. The Hb was 9.8 g.% and the W.B.C. 16,000 per c.mm. He failed to attend for follow-up.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 29

Patient J.D. African male Age 45 yrs. Occupation labourer

History: Six months of pleuritic pain in the right lower chest with a cough productive of thick white sputum. Associated symptoms were weight loss, anorexia and swelling of the feet. Past history of dysentery 6 months ago.

Examination: Apyrexial. Ill-looking. Emaciated. Pale mucosae. Ankle oedema. Finger clubbing.
Chest: Sputum, small quantities of thick yellow pus. Diminished movement, dullness on percussion and decreased air entry over the lower half of the right chest. Above this there was an area of hyperresonance. Localised intercostal tenderness in the 9th interspace at the mid-clavicular line.
Abdomen: Slight tenderness in the right upper quadrant with a tender liver enlarged 2 fingerbreadths below the right costal margin.

Investigations: Hb 8.8 g.%. W.B.C. 13,000 per c.mm. E.S.R. 51 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Large right-sided pleural effusion with an air-fluid level and patchy changes in the right lower lobe. Air-fluid levels were seen in the hepatic area and the presence of a subdiaphragmatic abscess cavity was postulated. The level of the right hemidiaphragm could not be ascertained.
Chest Aspirate: E.histolytica present. Gram-positive cocci and gram-negative bacteria seen on direct examination.
Bacterial Culture: Staphylococcus pyogenes and B.coli.
Sputum: E.histolytica not detected. Staphylococcus pyogenes cultured.
Steel: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 2nd day and postural drainage. Antibiotic therapy consisted of a course of penicillin and streptomycin followed by the broad-spectrum antibiotics tetracycline and penbritin. Transfused with 2 units of packed cells.

Progress: On the 2nd day 1,060 ml. of foul-smelling anchovy sauce pus was aspirated through the right 9th interspace. Bacterial examination yielded the results recorded above. Four further aspirations in the first 20 days brought the total fluid removed to 1500 ml. of pus. Subsequent cultures grew B.coli and Proteus vulgaris but no Staphylococci.

A low-grade pyrexia settled in the 3rd week and small quantities of purulent sputum were produced daily during the first month. Dullness and diminished air entry persisted at the right base.

Chest screening in the 3rd week showed a much elevated fixed right diaphragm with a large subdiaphragmatic abscess cavity. The right pleural effusion was much smaller and the air-fluid level no longer present.

A bronchogram showed collapse and bronchiectasis of the basal segments of the right lower lobe, with similar changes in the right middle lobe. No communication with the cavity below the right diaphragm was demonstrated.

Serial radiographs showed the cavity below the right diaphragm to have healed by the 6th week. At discharge the pleural effusion had completely resolved, there was residual elevation of the right hemidiaphragm and minimal adjacent right basal shadowing suggestive of pleural thickening and linear atelectasis.

At discharge on the 74th day the patient's weight had increased from 89 pounds to 115 pounds, dullness and diminished air entry persisted at the right base and the liver edge was no longer palpable. The Hb had risen to 15.7 g.%, the W.B.C. was 9,000 per c.mm. and the E.S.R. 14 mm. in one hour.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the diaphragm into the right pleural cavity from where it drained into a bronchus. Alternatively the liver abscess may initially have extended into the lung and then reached the right pleural cavity via a bronchopleural fistula.

Diagnosis: Amoebic liver abscess and an amoebic empyema with a bronchopleural fistula.

Case Report No. 30

Patient J.K. African male Age 21 yrs. Occupation labourer

History: Pleuritic pain in the right lower chest with a cough productive of large quantities of blood-stained sputum for 3 weeks. Associated symptoms were dyspnoea on exertion, tiredness and weight loss. No past history of dysentery.

Examination: Apyrexial. Ill-looking. Wasted. Pale mucosae. Finger clubbing.
Chest: Sputum, several hundred ml. of blood-stained pus coughed up on admission. Diminished movement, dullness on percussion and decreased air entry at the right base. Localised intercostal tenderness detected in the lower right rib spaces.
Abdomen: No right upper quadrant tenderness, the liver edge was not palpable.

Investigations: Hb 5.3 g.%. W.B.C. 11,000 per c.mm. E.S.R. 79 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Elevation of the right hemidiaphragm with consolidation of the middle lobe and patchy consolidation of the basal segments of the right lower lobe. A small interlobar effusion was present at the base of the greater fissure.
Sputum: Trophozoites of E.histolytica present.
Stool: Cysts of E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage.

Progress: Liver aspiration was attempted and failed. The patient remained apyrexial throughout his stay and blood-stained pus was coughed up in gradually decreasing quantities for the first 2 weeks.

Serial radiographs of the chest showed partial clearing of the right base with minimal residual patchy changes at discharge. The right diaphragm returned to its normal position with resolution of the interlobar effusion.

At discharge on the 22nd day there had been a weight increase of 8 pounds and dullness and diminished air entry persisted at the right base. The Hb had risen to 12 g.%, the W.B.C. was 5,000 per c.mm. and the E.S.R. 46 mm. in one hour.

At follow-up after one month the patient had improved further, there were no signs at the right base and the chest radiograph was normal.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended through the right diaphragm and drained into a bronchus.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula.

Case Report No. 31

Patient M.M. African male Age 60 yrs. Occupation unemployed

History: Pain with swelling in the right upper abdomen for 2 weeks. Two years previously treated for an amoebic liver abscess. No past history of dysentery.

Examination: Apyrexial. Wasted.
Chest: Trachea central. Dullness on percussion and diminished air entry at the right base.
Abdomen: A visible swelling in the right upper quadrant, the edge of which extended over the costal margin. The skin over this mass was distended, shiny and fluctuant with central pointing. A tender liver edge was palpated 8 fingerbreadths below the costal margin.

Investigations: Hb 12 g.%. W.B.C. 6,000 per c.mm. E.S.R. 51 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Elevation of the right diaphragm with a small pleural effusion in the right costophrenic angle and minimal adjacent shadowing suggestive of linear atelectasis in the basal segments of the right lower lobe.
Liver Aspirate: E.histolytica not detected. B.coli cultured from the 2nd aspirate.
Stool: E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 1st day. Oxytetracycline added on the 6th day.

Progress: The liver was aspirated on 1st and 5th days and a total of 450 ml. of blood-stained pus removed. Attempted aspiration of the pleural effusion failed. A swinging temperature followed the second aspiration, and because of threatened external rupture the secondarily infected amoebic abscess was drained surgically.

The temperature settled on the 10th day following surgery by which time the liver edge had receded to 2 fingerbreadths. Drainage ceased on the 15th day.

At discharge on the 26th day there had been a weight increase of 6 pounds and signs persisted at the right base. The Hb had risen to 14.2 g.% and the W.B.C. and E.S.R. were 7,000 per c.mm. and 18 mm. in one hour respectively. Radiographs showed a decrease in the pleural fluid with residual elevation of the right diaphragm. At follow-up after one month there were no signs at the right base and chest radiographs were normal.

Comments: The pleural effusion was probably of a presuppurative nature having resulted from involvement of the parietal pleura by the inflammatory reaction at the periphery of a secondarily infected amoebic liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pleural effusion.

Case Report No. 32

Patient T.M. African male Age 22 yrs. Occupation labourer

History: Pain in the right hypochondrium for 3 months, referred to the right shoulder. Three weeks prior to admission blood-stained sputum was coughed up and the haemoptysis continued until admission. No past history of dysentery.

Examination: Temperature 101°F. Ill-looking. Pale mucosae.
Chest: Coughing up anchovy sauce pus. Trachea central. Bulging of the right lower costal margin with intercostal tenderness in this region. Movement diminished, percussion note stony dull, air entry decreased and crepitations at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant and the liver edge 3 fingerbreadths below the costal margin.

Investigations: Hb 8.8 g.%. W.B.C. 15,000 per c.mm. E.S.R. 48 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Right diaphragm much elevated with a small area of consolidation in the adjacent lung field. On the right lateral view a cavity with an air-fluid level was seen in the right lower lobe and there was a small anteriorly situated effusion.
Liver Aspirate: E.histolytica not detected. Sterile on culture for pyogenic bacteria.
Pleural Aspirate: E.histolytica not detected. Acid-fast bacilli not detected. Sterile on culture for acid-fast bacilli and pyogenic organisms.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage. A 10-day course of tetracycline was included.

Progress: On admission 25 ml. of blood-streaked, yellow pus was aspirated from the liver through the 10th intercostal space. At a higher level 50 ml. of serous fluid was aspirated from the right pleural cavity.

The temperature settled on the 3rd day of treatment, sputum production ceased on the 5th day and abdominal tenderness and liver enlargement resolved by the 10th day. A further radiograph after 2 weeks showed a decrease in the level of the right diaphragm with resolution of the cavity in the right lower lobe and clearing of the effusion.

At discharge at the end of one month there had been a weight increase of 6 pounds and signs of an elevated right diaphragm persisted at the right base. Radiographs showed residual elevation of the right diaphragm with minimal shadowing in the adjacent lung field suggestive

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of pleural thickening.

Comments: The serous effusion probably resulted from involvement of the pleura by the inflammatory reaction at the periphery of either a liver abscess or a lung abscess.

Diagnosis: Amoebic liver abscess and an amoebic lung abscess with a presuppurative pleural effusion.

Case Report No. 33

Patient A.M. African male Age 29 yrs. Occupation labourer

History: Diarrhoea, blood and mucus for 2 weeks associated with pain in the right upper quadrant referred to the right shoulder, and a productive cough. Previous history of dysentery 9 years ago.

Examination: Temperature 102°F. Ill-looking.
Chest: Trachea central. Movement, percussion note and air entry were decreased at the right base and there was localised intercostal tenderness.
Abdomen: Tenderness and guarding in the right upper quadrant with the liver edge 2 fingerbreadths below the costal margin.

Investigations: Hb 13.6 g.%. W.B.C. 15,000 per c.mm. E.S.R. 28 mm. in one hour.
Chest Radiographs showed the right dome of the diaphragm to be obscured by a pleural effusion of moderate size.
Pleural Aspirate: 50 ml. of uniformly blood-stained serous fluid in which no amoeba organisms or acid-fast bacilli were seen or cultured.
Stool: Dysenteric. E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 2nd day.

Progress: The temperature settled and symptoms resolved by the 4th day. Right upper quadrant signs resolved in the 2nd week. There was no sputum coughed up during hospitalisation. Chest screening showed slight elevation of the right diaphragm with paradoxical movement. Liver aspiration was attempted unsuccessfully.

The patient was discharged free of symptoms but with dullness and diminished air entry persisting at the right base at the end of the 3rd week. There had been a weight increase of 10 pounds. The Hb at discharge was 13.4 g.% and the W.B.C. 9,000 per c.mm. Serial radiographs showed clearing of the fluid with residual elevation of the right diaphragm.

Comments: The serosanguineous effusion probably resulted from involvement of the pleura by the inflammatory reaction of the periphery of an amoebic liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pleural effusion.

Case Report No. 34

Patient S.M. African male Age 41 yrs. Occupation labourer

History: A continuous dull pain situated in the right upper abdomen for one month. Past history of dysentery with hospital admission 15 years ago.

Examination: Temperature 99°F. Pale mucosae.
Chest: Trachea central. Bulging of the right lower ribs with diminished movement, dullness on percussion and decreased air entry at the right base. Localised intercostal tenderness.
Abdomen: Tenderness in the right upper quadrant and the liver edge was not palpable.

Investigations: Hb 9.6 g.%. W.B.C. 17,000 per c.mm. E.S.R. 54 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Right diaphragm much elevated with a small pleural effusion at the right base.
Liver Aspirate: 540 ml. of anchovy sauce pus. Sterile on culture for bacteria, no E.histolytica found.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day.

Progress: A total of 660 ml. of anchovy sauce pus was obtained from the liver by aspiration on the 2nd and 9th days. The temperature settled on the 7th day and tenderness resolved on the 12th day after treatment with emetine and chloroquine.

Further radiographs on the 5th day showed, in addition to elevation, an air-fluid level below the right diaphragm, the air having been introduced at aspiration.

The patient was discharged on the 30th day symptom free with diminished percussion note and air entry at the right base and no signs in the right upper quadrant. There was a weight increase of 5 pounds. Radiographs at discharge showed the subdiaphragmatic cavity to be decreasing in size and the diaphragm to be less elevated with the small pleural effusion still present. The Hb had risen to 12.4 g.% and the W.B.C. was 5,000 per c.mm.

At follow-up one month later the patient was free of symptoms with minimal signs at the right base. Radiographs showed that the pleural effusion had resolved with slight elevation of the right diaphragm persisting.

Comments: The pleural effusion was probably of a presuppurative nature having resulted from involvement of the pleura by the inflammatory reaction at the periphery of an amoebic liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pleural effusion.

Case Report No. 35

Patient J.C. African male Age 45 yrs. Occupation labourer

History: Cough with pain in the right upper quadrant for 3 weeks. Associated symptoms were retrosternal pain with dyspnoea on exertion. There was no past history of dysentery.

Examination: Apyrexial. Wasted. Pale mucosae. Clubbing of the fingers. Chest: Trachea central. Diminished movement, stony dullness on percussion and decreased air entry at the right base with marked localised intercostal tenderness.
Abdomen: Guarding and tenderness in the right upper quadrant with the liver edge palpable 3 fingerbreadths below the right costal margin.
Cardiovascular System: Normal.

Investigations: Hb 9.3 g.%. W.B.C. 10,000 per c.mm. E.S.R. 60 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Moderate elevation of the right diaphragm with a small pleural effusion.
Stool: Dysenteric. E.histolytica not detected.
Liver Function Tests: Serum proteins: serum albumin 1.3 g.%, serum globulin 4.6 g.%, otherwise normal.

Treatment: Dehydroemetine, chloroquine and diodoquin from the 1st day.

Progress: Liver aspiration on the 3rd day yielded 1180 ml. of yellowish pus in which trophozoites of Entamoeba histolytica were found and which was sterile on culture for bacteria and acid-fast bacilli. On the 7th day 250 ml. of serous fluid was aspirated from the right chest which was sterile on culture, had a protein content of 3 g. and contained lymphocytes, a few polymorphs, histiocytes and mesothelial cells. The temperature which had risen to 101°F settled on the 8th day and symptoms cleared by the 10th day.

At discharge at the end of the second week the liver was no longer palpable, there were minimal signs at the right base and the patient had gained 5 pounds in weight. The Hb at discharge was 11.5 g.%, the W.B.C. 8,000 per c.mm., whilst the E.S.R. had fallen to 44 mm. in one hour. Serial radiographs showed the effusion to be decreasing with residual elevation of the right diaphragm.

Comments: The serous effusion probably resulted from involvement of the pleura by the inflammatory reaction at the periphery of an amoebic liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pleural effusion.

Case Report No. 36

Patient K.M. African male Age 28 yrs. Occupation labourer

History: Continuous pain of sudden onset in the right lower chest for 3 weeks, aggravated by coughing and breathing and associated with a cough productive of reddish-brown sputum. No past history of dysentery.

Examination: Temperature 102°F. Ill-looking. Evidence of recent weight loss. Pale mucosae.
Chest: Small quantities of purulent sputum. Trachea central. Stony dullness on percussion over the right chest with an overlying zone of hyperresonance and absent air entry. A succussion splash with a positive "coin test" heard at the right mid-zone. Intercostal tenderness found in lower right interspaces.
Abdomen: Tenderness and guarding in the right upper quadrant and a 3 fingerbreadths tender hepatomegaly.

Investigations: Hb 7.5 g.%. W.B.C. 14,000 per c.mm. E.S.R. 65 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: A large hydropneumothorax on the right with slight mediastinal shift. Position of the right diaphragm obscured by the overlying effusion.
Pleural Aspirate: Thick, greenish yellow, non-offensive pus. E.histolytica not detected. Sterile on culture for pyogenic bacteria and acid-fast bacilli.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day and crystalline penicillin one million units q.i.d. A 10-day course of tetracycline followed the penicillin. Transfusion with one pint of blood.

Progress: On admission 1350 ml. of thick greenish-yellow pus was aspirated from the right chest. During the first three weeks 14 aspirations were attempted and a total of 1915 ml. of pus removed. There was a gradual thinning of the pus which became dark brown in colour with successive aspirations. At no stage were pyogenic bacteria or acid-fast bacilli cultured from the aspirates.

Significant sputum production ceased after one week and right upper quadrant signs cleared in the second week. The temperature settled in the third week but signs at the right base persisted. Serial chest radiographs showed rapid clearing of the pleural fluid with absorption of the air in the first three weeks. The right diaphragm became visible and was seen to be elevated.

The patient was discharged free of symptoms on the 32nd day. Dullness and diminished air entry persisted over the right lung base. There had been a weight increase of 7 pounds. Chest radiographs

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showed complete clearing of the hydropneumothorax with re-expansion of the right lung and a thin rim of pleural thickening along its right lateral border. The Hb rose to 12.4 g.%, the W.B.C. was 9,000 per c.mm. and the E.S.R. 22 mm. in one hour.

Comments: It was postulated that an amoebic liver abscess eroded into the right pleural cavity with subsequent extension of the resulting empyema into a bronchus. Alternatively, the liver abscess may have extended into the right lung base with subsequent extension into the right pleural space.

Diagnosis: Amoebic liver abscess and an amoebic empyema with a broncho-pleural fistula.

Case Report No. 37

Patient M.N. African male Age 28 yrs. Occupation labourer

History: Cough with pain in the right chest and fever and weight loss for 6 months. Haemoptysis with purulent sputum for 4 months. No past history of dysentery. The patient had been attending a witch doctor.

Examination: Temperature 101°F. Ill-looking. Emaciated. Cold extremities. Pale mucosae.
Chest: Wasting of thoracic musculature maximal on the right. Small skin incisions made by a witch doctor were scattered over the right lower chest. Trachea central. Diminished movement, stony dullness and absent air entry over the right chest with localised intercostal tenderness at the right base.
Abdomen: Scaphoid with tenderness in the right upper quadrant and the liver not enlarged.

Investigations: Hb 7.6 g.%. W.B.C. 38,000 per c.mm. E.S.R. 76 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Massive right-sided effusion with no elevation of the right diaphragm and two air-fluid levels in the right upper zone. Shift of the mediastinum to left.
Pleural Aspirate: E.histolytica present, gram-negative bacilli cultured.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day. Terramycin administered intramuscularly for the first 2 days and then orally. Packed cells and plasma on admission and during the first week.

Progress: On admission 1200 ml. of thick anchovy sauce pus was aspirated from the right chest and the patient greatly relieved. Six hundred ml., 300 ml. and 360 ml. were aspirated on the 2nd, 3rd and 4th days respectively.

On the 4th day anchovy sauce pus was coughed up in large quantities and the patient developed signs of a pneumothorax at the right apex and there was deterioration in his general condition. A cannula was introduced into the 2nd interspace and air liberated through a water seal. Pus also drained through this tube.

On the 9th day the patient pulled out the drainage tube, his condition continued to deteriorate and he died.

Necropsy Findings: Approximately 700 ml. of blood-stained pus was found in the right pleural cavity. The right lung was collapsed and bound down by a greatly thickened visceral pleura.

A small abscess in the right lobe of the liver was found. This

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was situated posteriorly below the right diaphragm to which the superior surface of the liver was attached by adhesions. The parietal pleura overlying the abscess and the diaphragm in this region were thickened and adherent. There was a communication between the liver abscess and the right pleural cavity approximately 3 cm. in diameter. A bronchopleural fistula was not demonstrated and no abscesses were found in the right lung. Mucosal ulcers were found in the caecum and ascending colon.

Histology: Trophozoites of E.histolytica were identified in the wall of the liver abscess and in the margins of the ulcers in the large bowel.

Diagnosis: Amoebic liver abscess with an amoebic empyema and probable bronchopleural fistula.

Case Report No. 38

Patient M.C. African male Age 28 yrs. Occupation labourer

History: Pain had been experienced in the right lower thoracic region for 6 months associated with a cough productive of blood-stained, yellowish sputum. Six hours before admission the patient had a fit. Five further convulsions occurred en route to hospital. There was no past history of dysentery.

Examination: Apyrexial. Wasted. Comatose.
Central Nervous System: Comatose, responding to painful stimuli. Generalised convulsions observed. Neck rigidity. No papilloedema. No localising signs.
Chest: Dullness on percussion and absent air entry at the right base.
Abdomen: Nil of note detected.

Investigations: C.S.F.: A slightly turbid fluid with a pressure of 200 mm. Polymorphs 240, lymphocytes 86. Protein content 170 mgm.%. Chloride 700 mgm.%. Sugar 63 mgm.%. Direct examination showed no organisms and culture for pyogenic organisms and acid-fast bacilli was sterile.
Blood: Hb 12.3 g.%. W.B.C. 8,000 per c.mm.

Treatment: Penicillin, streptomycin and i.m. I.N.H. were started. No specific anti-amoebic therapy was given.

Progress: The level of consciousness deteriorated over the first 24 hours and the patient died on the 3rd day.

Necropsy Findings: The right pleural cavity contained 400 ml. of thick, yellow pus. There was gross thickening of the visceral and parietal pleura surrounding the empyema cavity at the base of the right lung. In parts these pleural layers measured up to 2 cm. in thickness.

The right diaphragm, the parietal pleura and the superior surface of the liver were bound together by dense adhesions. There was a small opening in the right diaphragm. Below this opening an area of scar tissue was seen in the superior aspect of the right lobe of the liver.

On opening the skull flattening of the cerebral convolutions was seen and a large brain abscess was found in the left parieto-occipital region.

Ulcers consistent with amoebic ulcers were found in the caecum.

Histology: E.histolytica were identified in the walls of the brain abscess and in the margins of the caecal ulcers.

Diagnosis: Amoebic colitis, amoebic liver abscess, amoebic empyema and an amoebic brain abscess.

Case Report No. 39

Patient F.M. African male Age 58 yrs. Occupation labourer

History: Cough with a pleuritic pain in the right lower chest for one week. Five days before admission approximately one cup of blood-stained sputum was coughed up. No past history of dysentery.

Examination: Temperature 101°F. Ill-looking. Wasted. Pale mucosae. Chest: Coughing up small quantities of dirty yellow blood-stained sputum. Signs of a large pleural effusion at the right base with tracheal and mediastinal shift and a pleural friction rub with crepitations in the right upper zone. Abdomen: Guarding and tenderness in the right upper quadrant and a tender liver edge palpated 3 fingerbreadths below the costal margin.

Investigations: Hb 8.7 g.%. W.B.C. 9,000 per c.mm. E.S.R. 62 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Large right sided effusion with tracheal and mediastinal shift. The level of the right diaphragm could not be ascertained.
Pleural Aspirate: E.histolytica not detected, on direct examination and culture there were no acid-fast bacilli or pyogenic organisms.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage.

Progress: On admission 500 ml. of anchovy sauce pus was aspirated from the right chest. Penicillin and streptomycin, followed by tetracycline, were added to the above treatment. A second course of 6 gr. of emetine was started in the 4th week.

There appeared to be an initial response to therapy but the temperature rose in the 2nd week and a low-grade pyrexia persisted for a further 8 weeks. The liver decreased in size and was no longer palpable in the 5th week. Small quantities of blood-stained sputum were produced daily for one month.

Serial radiographs of the chest showed rapid clearing of the effusion revealing elevation and irregularity of the right diaphragm with consolidation of the right middle and lower lobes and apparent cystic changes in these lobes. Bronchoscopy showed no abnormalities and permission for bronchography was refused.

Signs of crepitations and bronchial breathing at the right base persisted and were present at discharge during the 14th week. The weight increased by 12 pounds in hospital, the Hb rose to 14 g.%, the W.B.C. fell to 4,000 per c.mm. and the E.S.R. to 8 mm. in one hour.

At discharge on the 93rd day radiographs showed the right diaphragm to be in normal position and changes in the right lower and middle lobes which resembled those of cystic bronchiectasis.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which extended into the right pleural cavity and right lung base. The radiological changes suggestive of bronchiectasis were probably the residua of the amoebic lesion in the right lung.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula and an amoebic empyema.

Case Report No. 40

Patient Z.D. African male Age 40 yrs. Occupation labourer

History: A continuous vague abdominal pain associated with fever and vomiting for 4 days. No past history of dysentery.

Examination: Temperature 101°F.
Abdomen: Moderate tenderness in the epigastric region and left upper quadrant. The liver edge was not palpable.
Chest: Examination normal.

Investigations: Hb 11 g.%. W.B.C. 15,000 per c.mm. E.S.R. 52 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: No abnormality detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 4th day.

Progress: The temperature remained elevated and the symptoms and signs in the left upper quadrant increased in severity.

On the 4th day pain was experienced in the left chest and localised intercostal tenderness with signs of fluid were found at the left base. Radiographs at this stage showed a left pleural effusion aspiration of which yielded 30 ml. of thin, blood-stained fluid. This contained polymorphs and lymphocytes, no organisms were seen on direct examination or cultured and the protein content was 3.4 g.%. The clinical picture at this stage suggested an amoebic liver abscess and treatment with emetine and chloroquine was started.

A further aspiration of left chest on the 10th day yielded 400 ml. of typical anchovy sauce pus. Direct examination and culture of this aspirate revealed no amoeba, bacteria or acid-fast bacilli. On the 17th day a final 100 ml. of similar fluid was removed.

The temperature settled on the 16th day but pleuritic pain persisted until the 42nd day. Radiographs at this stage showed resolution of the pleural effusion with slight elevation of the left diaphragm, and minimal residual pleural thickening.

The patient was discharged on the 46th day free of symptoms with residual dullness and diminished air entry at the left base. He had gained 12 pounds in hospital and the final Hb was 14 g.%, the W.B.C. 9,000 per c.mm. and the E.S.R. 31 mm. in one hour.

Comments: The presuppurative effusion probably resulted from involvement of the left pleura by the inflammatory reaction at the periphery of a left lobe liver abscess. This abscess subsequently extended into the left pleural space producing an amoebic empyema.

Diagnosis: Left lobe amoebic liver abscess with an amoebic empyema.

Case Report No. 41

Patient D.M. African male Age 52 yrs. Occupation labourer

History: Pain in the right upper quadrant and lower chest associated with a swelling in the right upper quadrant for 6 weeks. The above symptoms had been associated with a productive cough and dyspnoea for 3 weeks. Past history of dysentery one year before admission.

Examination: Temperature 100°F. Ill-looking. Wasted. Pale mucosae.
Chest: No sputum coughed on admission. Trachea deviated to the left. Bulging of the right lower chest with diminished movement, stony dullness, absent air entry over the right chest and localised intercostal tenderness at the right base.
Abdomen: Diffuse swelling in the right upper quadrant with guarding and tenderness and the liver edge enlarged 4 fingerbreadths below the costal margin.

Investigations: Hb 10 g.%. W.B.C. 11,000 per c.mm. E.S.R. 52 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: The right diaphragm was elevated with an underlying air-fluid level and an area of circumscribed consolidation in the right lower lobe.
Sputum: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage. The antibiotics penicillin and streptomycin were given followed by tetracycline and chloromycetin.

Progress: The day after admission the patient collapsed and signs of shock were found with a blood pressure of 90/70 mm. of Hg. These were associated with signs of a large pleural effusion on the right side and mediastinal shift. Radiographs showed a massive right pleural effusion with an air-fluid level and collapse of the right lung. Aspiration of the right chest yielded 2280 ml. of greyish pus in which no amoeba were found. Gram-negative bacilli were detected in the aspirate and identified as Aerobacter aerogenes. These were sensitive to the tetracycline group, streptomycin and chloromycetin. Tetracycline was added to therapy.

On the 3rd day 450 ml. of anchovy sauce pus was coughed up and it was considered that following rupture of the abscess into the right pleural cavity a bronchopleural fistula had developed.

On admission it was not realised that the patient had already received 5 grains of emetine. This resulted in the administration of 15 gr. of emetine over a period of 17 days. This large quantity was considered to be a possible explanation of two episodes of paroxysmal ventricular tachycardia which occurred during the first month.

For the first 6 weeks a low-grade pyrexia persisted and during this period the chest was aspirated 17 times yielding a total of 10,950 ml.

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of pus. Aerobacter aerogenes was continually cultured and became increasingly resistant to broad-spectrum antibiotics. Further radiographs showed fairly rapid accumulation of fluid following aspiration. Tube drainage of the empyema was instituted at 6 weeks and for a further 6 weeks the quantity of pus drained gradually diminished.

At 12 weeks a low-grade pyrexia persisted, the intercostal tube was removed and the patient transferred to a surgical ward for consideration of decortication of a right lung which was collapsed and bound down by a thickened pleura.

During the 14th week he developed pain and tenderness in the epigastrium, his condition deteriorated and 150 ml. of greenish pus was aspirated from the left lobe of the liver. Two days later the patient died.

Necropsy Findings: Approximately 2 pints of pus were removed from a large empyema cavity in the right chest. There was gross thickening of the pleura surrounding the empyema cavity with collapse and congestion of the adjacent lung. The peritoneal cavity contained pus. The superior surface of the liver, the right diaphragm and the parietal pleura were bound together by dense adhesions. A focus of scar tissue thought to represent a healed liver abscess was found in the upper aspect of the right lobe of the liver. There was no evidence of a communication between the empyema cavity and the subphrenic space. An abscess approximately 12 cms. in diameter was found in the left lobe of the liver. Multiple small abscesses were found in the kidneys. There was no evidence of ulceration of the large bowel.

Histology: No amoeba were found in the walls of the liver abscess or the empyema cavity. The multiple small abscesses in the kidney were of pyogenic origin.

Diagnosis: Amoebic liver abscesses and an amoebic empyema with a bronchopleural fistula.

Case Report No. 42

Patient M.M. African male Age 48 yrs. Occupation labourer

History: Sudden onset of pain in the right lower chest 16 days before admission associated with a bursting sensation in the right upper abdomen. The pain was pleuritic in nature and radiated to the right shoulder. Associated symptoms were cough productive of yellow sputum and dyspnoea. No past history of dysentery.

Examination: Temperature 99°F. Ill-looking. Wasted. Pale mucosae. Chest: Small quantities of purulent sputum. Trachea deviated to the left. Decreased movement, stony dullness, absent air entry over the right chest with localised intercostal tenderness at the right base. Abdomen: Tenderness and guarding in the right upper quadrant with a 3 fingerbreadths tender enlarged liver.

Investigations: Hb 10.4 g.%. W.B.C. 6,000 per c.mm. E.S.R. 56 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Sputum: E.histolytica not detected.
Chest Radiographs: Showed a massive right-sided pleural effusion. There was mediastinal shift and an air-fluid level at the right apex thought to be due to air introduced at aspiration. Forty ml. of lipiodol were introduced at the second aspiration and screening showed the right diaphragm to be moderately raised but did not demonstrate an hepatopleural fistula.
Stools: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 3rd day. Breathing exercises started in the second week.

Progress: On admission 2,800 ml. of typical anchovy sauce pus was aspirated from the right chest. Entamoeba histolytica were isolated on direct examination and the fluid was bacteriologically sterile. In the first 3 weeks 10 aspirations were performed and a total of 4,710 ml. of fluid was removed. This remained sterile and gradually became serous in character.

The temperature settled on the 3rd day, pain and tenderness were absent and sputum production ceased after one week. At discharge after 4 weeks there was still signs at the right lower half of the chest and the liver was no longer palpable. The Hb rose to 13.4 g.% and the W.B.C. was 6,000 per c.mm. Radiographs at this stage showed translucent lung at the right base with an opaque shadow above the translucency involving nearly all the right hemithorax which was thought to represent pleural thickening.

Two months after discharge the patient was symptom free and had gained 12 pounds in weight. Air entry at the right base had

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improved and there was no effort dyspnoea. Bronchography at this stage showed distortion of the bronchial tree on the right with shift of the right main bronchus to the left, crowding of lower and middle lobe bronchi and absence of filling of right upper lobe bronchi. Stenosis of right upper main bronchus was postulated but bronchoscopy showed this orifice to be patent.

Six months after discharge the patient remained well and was doing a full day's work with further improvement of air entry in the right chest. Eighteen months after discharge the above progress was maintained and there had been a further weight increase of 23 pounds. Radiographic changes, interpreted as those of pleural thickening, persisted in the right chest.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the right pleural cavity. The residual pleural thickening was probably the result of inadequate drainage of the empyema.

Diagnosis: Amoebic liver abscess and an amoebic empyema with residual pleural thickening.

Case Report No. 43

Patient E.D. African male Age 44 yrs. Occupation labourer

History: Pain in the right lower chest radiating to the right shoulder with associated cough and dyspnoea for 8 weeks. Pain in the right upper quadrant with diarrhoea, blood and mucus during the above period.

Examination: Temperature 102°F. Ill-looking. Wasted. Pale mucosae. Pulse rate 108 per minute with small volume pulse.
Chest: Bulging of the right lower ribs with diminished movement, dullness on percussion and absent air entry over the right lower half of the chest. Intercostal tenderness at the right base.
Abdomen: Guarding and tenderness in the right upper quadrant and a tender liver edge palpated 4 fingerbreadths below the costal margin.

Investigations: Hb 11.2 g.%. W.B.C. 17,000 per c.mm. E.S.R. 53 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Showed a much elevated right diaphragm with a pleural reaction at the right base.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day. Tetracycline from the 5th day.

Progress: On the 4th day there was a sudden exacerbation of pain in the right lower chest and signs of peripheral vascular collapse. The trachea and apex beat were displaced to the left and stony dullness and absent air entry were found at the right base. It was postulated that a liver abscess had ruptured into the right pleural space. Aspiration of the right chest yielded 1,800 ml. of yellow pus in which no amoeba were found and which was sterile on culture for pyogenic bacteria.

The patient's condition continued to deteriorate and he died on the 6th day.

Necropsy Findings: The right pleural cavity contained approximately 800 ml. of thin yellow pus. There was an opening in the posterior aspect of the right diaphragm which admitted 2 fingers and communicated with a large posteriorly situated liver abscess.

There was extensive ulceration of the caecum, ascending and sigmoid colon. Histology showed the liver abscess and intestinal lesions to be amoebic in origin.

Diagnosis: Amoebic dysentery, amoebic liver abscess and an amoebic empyema.

Case Report No. 44

Patient M.C. African male Age 34 yrs. Occupation labourer

History: Cough productive of approximately one cupful of blood-stained yellow sputum daily for 2 weeks. Pleuritic pain of sudden onset in the right chest for one week with dyspnoea on exertion. No past history of dysentery.

Examination: Temperature 99°F. Pale mucosae.
Chest: No sputum coughed. Trachea shifted to the left. Diminished movement, stony dull percussion note and absent air entry over the right lower chest with intercostal tenderness in the right lower rib spaces.
Abdomen: Tenderness and guarding in the right upper quadrant with a tender liver edge palpated 2 fingerbreadths below the right costal margin.

Investigations: Hb 10 g.%. W.B.C. 11,000 per c.mm. E.S.R. 47 mm. in one hour. Peripheral smear: Normochromic normocytic anaemia.
Chest Radiographs: Massive right sided pleural effusion (possibly encysted) with mediastinal shift. The position of the right diaphragm was obscured by the effusion.
Chest Aspirate: Anchovy sauce pus. E.histolytica present. Sterile on culture for pyogenic and acid-fast bacilli.

Treatment: Emetine, chloroquine and diodoquin from the first day.

Progress: On admission 450 ml. of anchovy sauce pus was aspirated from the right lower chest. Nine aspirations in the first month yielded a total of 3690 ml. of pus. The aspirate gradually thinned and assumed a dark brown colour. At no stage were pyogenic or acid-fast bacilli cultured. After the second week no further fluid was obtained from the right base whereas aspiration of the right upper chest continued to yield large quantities.

The temperature settled on the 4th day and right upper quadrant signs and symptoms in the first week. Signs of the right sided effusion persisted.

Serial radiographs in the first month showed a progressive decrease in the size of the effusion and indicated that in addition to the basal effusion an encysted effusion occupied the right upper lung field. The right diaphragm was not elevated.

The patient was discharged on the 42nd day free from symptoms having gained 8 pounds during hospitalisation. Percussion dullness and decreased air entry persisted at the right base and in the right upper axilla. Radiographs showed a small residual right basal effusion with a small encysted effusion in the right axillary region. The Hb rose to 13 g.% and the W.B.C. was 11,000 per c.mm.

After one month the encysted effusion had increased in size. Drainage was performed via an intercostal catheter and a further course of emetine and chloroquine was administered. At discharge after a further month in hospital the encysted effusion had cleared completely.

Comments: The clinical findings were interpreted as being those of an amoebic empyema which had resulted from extension of an amoebic liver abscess through the right diaphragm.

Diagnosis: Amoebic liver abscess and an amoebic empyema.

Case Report No. 45

Patient A.S. African male Age 26 years Occupation labourer

History: Stabbing pain in the right lower chest and dyspnoea for 7 weeks. Cough productive of reddish brown sputum for 5 weeks. Four weeks before admission the patient was treated for pneumonia with no response in an outlying hospital. No past history of dysentery.

Examination: Temperature 100°F. Ill-looking. Pale mucosae.
Chest: Coughing up anchovy sauce sputum. Trachea and apex beat deviated to the left. Diminished movement, dullness on percussion, and absent air entry over the lower half of the right chest. Localised intercostal tenderness at the right base.
Abdomen: No tenderness. Liver not enlarged.

Investigations: Hb 9.3 g.%. W.B.C. 18,000 per c.mm. E.S.R. 65 mm. in one hour. Peripheral smear: Normochromic normocytic anaemia.
Chest Radiograph: Large posteriorly situated encysted effusion in the right chest, deviation of the mediastinum to the left, and elevation of the right hemidiaphragm.
Sputum: E.histolytica present.
Pleural Aspirate: Blood-stained pus. E.histolytica present. Sterile on culture for pyogenic and acid-fast bacteria.

Treatment: Emetine, chloroquine and diodoquin, and postural drainage from the 2nd day.

Progress: Five hundred ml. of blood-stained pus was aspirated from the right chest on the first day and a further 100 ml. on the third day. Trophozoites of E.histolytica were seen on direct examination of the second aspirate and in the sputum.

The temperature settled on the 6th day and symptoms within the first week. Significant sputum production ceased in the second week.

At discharge in the fourth week the patient had gained 8 pounds, was symptom free and there had been considerable clearing of the signs in the right chest. The Hb was 13.6 g.% and the W.B.C. 8,000 per c.mm. Radiographs showed resolution of the pleural effusion, thickening of the greater fissure and shadows at the right base suggestive of pleural thickening. There was persisting elevation of the right hemidiaphragm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had ruptured into the right lung base and the right pleural cavity.

Diagnosis: Amoebic liver abscess with an hepatobronchial fistula and an amoebic empyema.

Case Report No. 46

Patient S.B. African male Age 36 yrs. Occupation labourer

History: Stabbing pain involving the right lower chest for 8 weeks associated with cough and dyspnoea. There was a past history of dysentery 8 weeks before admission.

Examination: Temperature 99°F. Wasted.
Chest: Trachea and apex beat displaced to the left. Diminished movement, stony dullness on percussion and impaired air entry over nearly the whole of the right chest.
Abdomen: Tenderness and guarding in the right upper quadrant. Liver edge not palpable.

Investigations: Hb 11.6 g.%. W.B.C. 11,000 per c.mm. E.S.R. 50 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Massive right-sided pleural effusion with mediastinal shift. The level of the right diaphragm was obscured by the effusion.
Pleural Aspirate: E.histolytica present. Sterile on culture for pyogenic organisms and acid-fast bacilli.

Treatment: Dehydroemetine, chloroquine and diodoquin from the 1st day. Breathing exercises were started during the 2nd week.

Progress: Two litres of thin anchovy sauce pus containing E.histolytica was aspirated from the right chest on admission. Five subsequent aspirations increased the volume of pus removed to 6980 ml. All specimens were sterile on culture for pyogenic organisms and acid-fast bacilli. With repeated aspirations the pus gradually changed to a serous fluid.

The temperature settled on the 3rd day and the abdominal symptoms and signs cleared by the 10th day. Serial radiographs showed the effusion at the right base to be decreasing in size. The patient discharged himself from hospital in the 4th week.

At follow-up after one month there were no symptoms but signs persisted at the right base. The weight increase of 10 pounds in hospital had been maintained. Radiographs showed a further decrease in the size of the effusion with changes suggestive of pleural thickening and the right diaphragm was in normal position.

Comment: The clinical findings were interpreted as being those of an amoebic empyema following extension of an amoebic liver abscess into the right chest.

Diagnosis: Amoebic liver abscess and an amoebic empyema.

Case Report No. 47

Patient M.V. African male Age 41 yrs. Occupation labourer

History: Pain in the right lower chest and right upper quadrant for one year. The pain radiated to the right shoulder tip and was aggravated by a non-productive cough. Several cupfuls of blood-stained sputum were suddenly coughed up 2 months before admission, followed by the continuous production of small quantities of blood-stained yellow sputum. No past history of dysentery.

Examination: Apyrexial. Wasted. Pale mucosae.
Chest: Coughing up blood-stained pus. Diminished movement, stony dullness and absent air entry over the right chest with localised intercostal tenderness at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant with a 2 fingerbreadths tender enlarged liver.

Investigations: Hb 7 g.%. W.B.C. 11,000 per c.mm. E.S.R. 68 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Right diaphragm not visible. There was a large posteriorly situated encysted effusion with an air-fluid level and shift of the mediastinum to the right suggesting collapse of the right lower lobe.
Sputum: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and postural drainage. Transfused with 2 units of packed cells.

Progress: The patient was apyrexial throughout his stay and continued to cough decreasing amounts of blood-stained sputum for 2 weeks. Chest aspiration and liver aspiration were attempted without success.

At discharge at the end of the 4th week he was symptom free, had gained 10 pounds, the liver was not enlarged and signs of fluid persisted at the right base. The Hb had risen to 11.5 g.%. The W.B.C. was 6,000 per c.mm. Chest radiographs at this stage showed a decrease in the fluid with an increase in the air above the fluid level. The diaphragm was moderately elevated.

At follow-up clinic at one and 4 months, progress was maintained but signs persisted at the right base. Chest radiographs showed a small residual effusion with no air in the pleural cavity and residual elevation of the right diaphragm.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which had extended into the right pleural cavity

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with subsequent drainage of the resultant empyema via a broncho-pleural fistula.

Diagnosis: Amoebic liver abscess and an amoebic empyema with a bronchopleural fistula.

Case Report No. 48

Patient N.D. African male Age 35 yrs. Occupation labourer

History: Abdominal pain, low backache and diarrhoea with blood and mucus for 3 days. More than 10 stools were passed daily. No past history of dysentery.

Examination: Temperature 100°F. Ill-looking. Moderate dehydration. Emaciated.
Chest: Examination normal.
Abdomen: Generalised tenderness and guarding with no distension and audible bowel sounds. The liver edge was not palpable.

Investigations: Hb 12.5 g.%. W.B.C. 46,000 per c.mm. E.S.R. 56 mm. in one hour.
Stool: Dysenteric. E.histolytica present.

Treatment: Emetine, chloroquine and diodoquin from the 1st day. Oral therapy was stopped on the 3rd day and intravenous fluids with gastric suction and intravenous tetracycline substituted.

Progress: On the 3rd day abdominal distension with signs of peritonitis and ileus developed. Perforation of an amoebic colitis into the peritoneum was diagnosed.

On the 12th day a tender, enlarged liver was palpated and a total of 900 ml. of anchovy sauce pus was subsequently aspirated from an area of localised tenderness in the right upper quadrant. All specimens were sterile on culture and no amoeba were found.

On the 15th day signs suggestive of a pleural effusion with a pleural friction rub were found at the right base. Chest radiographs showed elevation of the right diaphragm with adjacent patchy consolidation and a small costophrenic effusion. Aspiration through the right lower rib spaces yielded 100 ml. of serosanguineous fluid in which no amoeba were found and which when cultured was free of bacteria. A subsequent aspiration at the same site yielded 300 ml. of anchovy sauce pus. It was postulated that these findings represented a small serosanguineous effusion resulting from an adjacent subdiaphragmatic abscess which subsequently ruptured into the pleural cavity. Serial radiographs and a bronchogram obtained during the following 8 months showed the effusion to have cleared after 2 weeks, but minimal diaphragmatic elevation and costophrenic shadowing suggestive of pleural thickening persisted.

The patient was hospitalised for a total of 9 months as a result of the following associated complications. In the 8th week he developed an injection abscess in the right buttock following a second

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course of emetine. In the 11th week his general condition remained poor, pain was experienced in the left upper quadrant and a tender mass developed in this situation. Radiographs showed elevation of the left diaphragm with a small effusion. A left-sided sub-diaphragmatic abscess was diagnosed. Prior to aspiration of this abscess the patient suddenly collapsed, the mass disappeared and signs of a generalised peritonitis with ileus developed. Rupture of a left lobe liver abscess into the peritoneal cavity was diagnosed. In view of the patient's critical condition the surgeons advised conservative treatment. Gastric suction and intravenous therapy was continued for 10 days, transfusions of blood and plasma were given and broad-spectrum antibiotics used. Six aspirations of the left subdiaphragmatic region from the 11th to the 20th week yielded 720 ml. of foul-smelling pus in which no amoeba were found but from which B.coli were cultured on 3 occasions. Radiographs during this period showed the left-sided effusion to have cleared after 2 weeks.

In the 21st week progress was further retarded by a further episode of dysentery.

In the 26th week right lower chest pain was experienced and radiographs showed right lower lobe consolidation to be present. This episode of pneumonia or possible pulmonary infarction cleared radiologically within one month. The etiology was not established.

The patient's general condition gradually improved from the 30th week. Broad-spectrum antibiotics, blood and plasma transfusions with high protein diet and aspiration of abscesses formed the basis of treatment. Surgical drainage of a secondarily infected left sub-diaphragmatic abscess was refused by the patient. A low-grade pyrexia finally settled in the 24th week and the elevated W.B.C. in the 36th week. The Hb rose to 17 g.%. There was a weight increase of 40 pounds starting at 79 pounds and rising to 119 pounds. He was discharged during the 37th week with no residual chest or abdominal signs and a normal chest radiograph except for minimal right basal pleural thickening. After 3 months clinical and radiological progress was maintained.

Diagnosis: Amoebic colitis with perforation and peritonitis. Multiple amoebic liver abscesses. Amoebic pleural effusions and amoebic empyema. Rupture of a secondarily infected left lobe liver abscess with peritonitis.

Case Report No. 49

Patient L.M. African male Age 40 yrs. Occupation labourer

History: Diarrhoea, blood and mucus with abdominal pain for 6 months. Five days before admission pain was experienced in the right lower chest and a large quantity of blood-stained pus was coughed up. Past history of dysentery 4 years ago.

Examination: Temperature 99°F. Ill-looking. Emaciated. Pale mucosae. Chest: Sputum, blood-stained pus. Diminished movement, stony dullness on percussion and absent air entry at the right base.
Abdomen: Tenderness and guarding in the right upper quadrant and a tender enlarged liver palpated 3 fingerbreadths below the right costal margin.

Investigations: Hb 6.3 g.%. W.B.C. 6,000 per c.mm. E.S.R. 68 mm. in one hour. Peripheral smear: Normocytic normochromic anaemia.
Chest Radiographs: Elevation of the right diaphragm with an adjacent area of consolidation; a small effusion at the base of the right greater fissure and a posterior effusion on the right of moderate size.
Chest Aspirate: E.histolytica not detected, pyogenic bacteria and acid-fast bacilli not detected or cultured.
Stool: Dysenteric. Trophozoites of E.histolytica present.

Treatment: Emetine, chloroquine, diodoquin and tetracycline from the 3rd day and postural drainage.

Progress: The day of admission 500 ml. of reddish pus was aspirated from the right chest. A further 300 ml. of yellowish-brown, thin pus was aspirated from the same site on the 9th day and lipiodol introduced at aspiration showed that the pus was being removed from above the diaphragm.

The temperature settled on the 5th day but approximately 250 to 500 ml. of blood-stained pus was coughed daily until the 8th day. Signs of fluid persisted at the right base and a pleural rub developed following aspiration. The liver decreased in size and its firmness suggested associated cirrhosis. Liver function tests were also suggestive of cirrhosis and paracentesis abdominus yielded 10 ml. of a serous fluid with a protein content of 2.8 g. which contained a few lymphocytes but was sterile on culture for pyogenic and acid-fast bacilli.

The patient was discharged at the end of the 7th week free from symptoms with minimal signs at the right base. He gained 11 pounds in hospital. The Hb was 9.8 g.%, the W.B.C. 4,000 per c.mm. Radiographs at discharge showed almost complete absorption of fluid at the right base, a small effusion at the base of the greater fissure and shadows suggestive of residual pleural thickening. The area of consolidation had resolved and the right diaphragm was slightly elevated.

Comments: The clinical findings were interpreted as being those of amoebic colitis and an amoebic liver abscess which had extended into the right lung base and the right pleural cavity.

Diagnosis: Amoebic colitis, amoebic liver abscess with an hepato-bronchial fistula and an amoebic empyema.

Case Report No. 50

Patient M.S. African male Age 22 yrs. Occupation labourer

History: Cough with pleuritic pain in the right lower chest for 4 months. Haemoptysis for one week followed by a cough productive of dirty yellow sputum for 3 weeks. Associated symptoms were dyspnoea on exertion and swelling of the feet. No past history of dysentery.

Examination: Apyrexial. Wasted. Ankle oedema. Pale mucosae.
Chest: Sputum, small quantities of yellow pus. Trachea and apex beat shifted to the left. Diminished movement, stony dullness on percussion and absent air entry over the whole of the right chest, the right apex excluded. Crepitations and a pleural rub heard over the right chest.
Abdomen: Slightly distended, with tenderness, guarding and a 2 fingerbreadths tender enlarged liver.

Investigations: Hb 6.3 g.%. W.B.C. 39,000 per c.mm. E.S.R. 65 mm. in one hour. M.C.H.C. 28%. Peripheral smear: Normocytic hypochromic anaemia.
Chest Radiographs: Massive right sided encysted effusion with slight shift of the mediastinum to the left. The position of the right diaphragm was obscured by the effusion.
Pleural Aspirate: Trophozoites of E.histolytica present.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the 2nd day. A 10-day course of tetracycline and transfusion with 2 units of packed cells and postural drainage were included in the treatment.

Progress: The right chest was aspirated 6 times in the first 2 weeks and a total of 2,490 ml. of pus removed. The initial aspirate was a greyish-yellow, thick pus, but the character gradually changed to a thinner blood-stained fluid with debris. Trophozoites of Entamoeba histolytica were found in 3 specimens and all specimens were sterile on culture for bacteria and acid-fast bacilli.

A large quantity of blood-stained purulent sputum was coughed on the 4th day and decreased quantities for the next 3 weeks. No amoeba, acid-fast bacteria or bacteria were found in sputum specimens. A low-grade pyrexia settled after one week, percussion note and air entry gradually improved at the right base and the liver edge was no longer palpable in the 3rd week.

Serial radiographs showed a progressive decrease in the size of the effusion and a pyopneumothorax. The right diaphragm was elevated and patchy changes suggestive of atelectasis and consolidation were present in the right lower lobe. Later films showed complete absorption of fluid and air with patchy changes in the right lower lobe suggestive of pleural thickening. Chest screening showed the right diaphragm to be elevated and immobile.

At discharge on the 31st day the patient was free of symptoms, he had gained 10 pounds in weight and there was minimal residual dullness and diminished air entry at the right lung base. The Hb had risen to 10 g.%, the W.B.C. was 10,000 per c.mm. and the E.S.R. 45 mm. in one hour.

After one month the patient remained well, there were no symptoms but slight dullness and diminished air entry persisted at the right lung base. Radiographs showed further clearing of the patchy consolidation, minimal elevation of the right diaphragm, thickening of the greater fissure and patchy shadowing suggestive of pleural thickening at the right base.

Comments: The clinical findings were interpreted as being those of an amoebic liver abscess which extended into the right pleural cavity with subsequent drainage of the empyema via a bronchopleural fistula.

Diagnosis: Amoebic liver abscess and an amoebic empyema with a bronchopleural fistula.

APPENDIX B

CASE REPORTS OF 15 PATIENTS WITH PERICARDIAL AMOEBIASIS

Case Report No. 51

Patient J.N. African male Age 17 yrs. Occupation labourer

History: Retrosternal pain and pain over the right lower chest for 5 weeks. Associated symptoms were increasing dyspnoea, a non-productive cough, fever and sweating. No past history of dysentery.

Examination: Temperature subnormal. Ill patient. Dyspnoeic. Pale mucosae. Cold extremities.
C.V.S.: Pulse rate 130 per minute, pulse volume small and pulsus paradoxus detected. Jugular venous pressure raised to the angle of the jaw. Blood pressure 110/80 mm Hg. The outer limit of cardiac dullness was percussed at the anterior axillary line and the apex beat was not palpable. Auscultation revealed a gallop rhythm with a pericardial friction rub.
Chest: Signs suggestive of an elevated right diaphragm or pleural effusion at the right base.
Abdomen: Visible epigastric swelling localised to the left of the midline. Tender liver edge 3 fingerbreadths enlarged with maximal tenderness over the epigastric swelling.

Investigations: Hb 7.3 g.%. W.B.C. 46,000 per c.mm. E.S.R. 62 mm. in one hour.
Chest Radiographs: Enlarged heart with a globular outline and a cardiothoracic ratio of 70%. Normal diaphragmatic position and lung fields.
Chest Screening: Decreased cardiac pulsation. Normal diaphragmatic movement.
E.C.G.: Elevated S-T segments in the standard leads, aVF and the chest leads V2 - V6.
Stool: Dysenteric. E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day.

Progress: On admission three attempts at aspiration of the epigastric mass failed and laparotomy revealed an enlarged liver with its anterior surface adherent to the abdominal wall. A left lobe liver abscess was aspirated and 180 ml. of anchovy sauce like pus removed.

On the 14th day pericardial aspiration yielded 90 ml. of heavily blood-stained serous fluid in which no amoeba, bacteria or acid-fast bacilli were seen and which was sterile on culture. Further radiographs showed that small bilateral effusions had developed and that the heart size was decreasing.

The liver edge and epigastric mass were no longer palpable after the 2nd week. The temperature settled in the 3rd week and by the 4th week no clinical signs of pericardial involvement remained. Serial

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E.C.G.'s showed return of the S-T segments to the iso-electric line with inversion of the T waves in the standard, unipolar and chest leads.

At discharge in the 9th week there was no evidence of cardiac disease, radiographs showed the transverse diameter of the heart to be within normal limits and small bilateral effusions.

Three weeks after discharge the patient was readmitted with symptoms and signs of a right-sided liver abscess. Chest radiographs showed elevation of the right diaphragm with an increase in size of the right effusion. A localised swelling developed over the laparotomy scar in the epigastrium, this ruptured and discharged anchovy sauce like pus in which E.histolytica were found. Further courses of emetine and chlorequine with penicillin and streptomycin were given. Response was satisfactory. At discharge 22 days after readmission there had been a weight increase of 12 pounds, the Hb was 16.2 g.%, the white cell count 10,000 per c.mm. and the E.S.R. 21 mm. in one hour.

At follow-up after 15 months the patient remained well, there were no cardiac, respiratory, or abdominal signs. Radiographs showed the right diaphragm to be in normal position with a minimal adjacent pleural reaction. The heart size was normal. Serial electrocardiographs during the above period showed the inverted T waves had become upright.

Comments: The clinical findings were interpreted as being those of a presuppurative pericardial effusion resulting from involvement of the pericardium by the inflammatory reaction at the periphery of a left lobe liver abscess.

Diagnosis: Amoebic liver abscesses with a presuppurative pericardial effusion.

Case Report No. 52.

Patient R.S. African male Age 40 yrs. Occupation labourer

History: Cough with pleuritic pain in the right lower chest for 3 days. Severe retrosternal pain with dyspnoea on exertion for 2 days. No past history of dysentery.

Examination: Temperature 99°F. Ill-looking. Pale mucosae. Ankle oedema.
C.V.S.: Small volume pulse with a rate of 96 per minute and pulsus paradoxus. The jugular venous pressure was not visibly raised. Blood pressure 90/60 mm Hg. The area of cardiac dullness was increased to the left and the apex beat not palpable. The heart sounds were distant and there was a loud pericardial friction rub.
Chest: Dullness on percussion and diminished air entry at the right base.
Abdomen: Liver edge 3 fingerbreadths enlarged and non tender with minimal epigastric tenderness.

Investigations: Hb 11.6 g.%. W.B.C. 64,000 per c.mm. E.S.R.: 52 mm. in one hour.
Chest Radiographs: Marked elevation of the right dome of the diaphragm with shadowing suggestive of segmental atelectasis of the adjacent right lower lobe and a small right pleural effusion. The heart was not enlarged but was displaced to the left. Radiographs of the abdomen showed the stomach to be displaced downwards and to the left by an epigastric mass.
E.C.G.: Low voltage with flattened T waves in the Std. and unipolar limb leads and inverted T waves throughout the chest leads.
Liver Aspirate: E.histolytica not detected. Sterile on culture for bacteria and acid-fast bacilli.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day. Terramycin was added to the above treatment.

Progress: On admission 200 ml. of thick, blood-stained pus was aspirated through the left upper epigastrium from the left lobe of the liver. A further 2 attempts to obtain pus from this site with one attempt at pericardial aspiration failed. In view of the elevated right diaphragm, aspiration of the right lobe of the liver through the 10th interspace was attempted on the 3rd day and failed. Repeat attempts on the 12th and 17th days yielded a total of 880 ml. of sterile anchovy sauce pus from a right lobe abscess. Amoeba were not identified in these aspirates.

A low-grade pyrexia settled at the end of the 3rd week. Signs of pericarditis resolved in the 3rd week and at this time a pleural friction rub was heard at the left base.

Serial radiographs showed a decrease in the level of the elevated right diaphragm and clearing of the right-sided effusion. They also showed an elevated left diaphragm with a small left basal effusion. Radiologically the transverse diameter of the heart remained within normal limits with no significant alteration of cardiac contour. Chest screening showed normal cardiac pulsation and immobility of both diaphragms.

An E.C.G. at one month showed generalised T wave inversion to have persisted.

The patient was discharged in the 7th week, free of symptoms with no cardiac signs but signs of residual elevation of the right diaphragm persisted. There was a weight increase of 6 pounds during hospitalisation. The Hb at discharge was 11.4 g.%, the white cell count 10,000 per c.mm. and the E.S.R. 54 mm. in one hour. Chest radiographs at this time showed residual elevation of the right diaphragm with shadowing suggestive of minimal thickening of the adjacent pleura. There was failure to attend follow-up after one month.

Comments: The clinical findings were interpreted as being those of pericarditis resulting from pericardial involvement by the inflammatory reaction at the periphery of a left lobe liver abscess.

Diagnosis: Amoebic liver abscesses with a presuppurative pericardial effusion.

Case Report No. 53

Patient S.M. African male Age 22 yrs. Occupation labourer

History: Continuous pain in the epigastrium radiating to the left neck and shoulder for 3 weeks. Associated symptoms were dyspnoea on exertion, a non-productive cough and pain in the left axilla. No past history of dysentery.

Examination: Temperature 103°F. Ill-looking. Wasted. Dyspnoeic. Pale mucosae.
C.V.S.: Small volume pulse with a rate of 140 and pulsus paradoxus. Jugular venous pressure raised to the angle of the jaw. Blood pressure 105/65 mm of Hg. The apex beat was not palpable and a pericardial friction rub was heard maximally over the 4th left interspace.
Chest: Normal.
Abdomen: 2 fingerbreadths enlarged tender liver.

Investigations: Hb 10.4 g.%. W.B.C. 29,000 per c.mm. E.S.R. 63 mm. in one hour.
Chest Radiographs: Slight increase in transverse diameter of the heart with loss of the normal cardiac contour.
Chest Screening: Normal cardiac pulsation. Normal diaphragmatic movement.
E.C.G: S-T segment elevation was present in standard lead I, aVL, and leads V1 - V6.
Stool: E.histolytica not seen.

Treatment: Streptomycin, izoniasid with emetine and chloroquine from the 1st day. The patient was digitalised and mersalyl given twice weekly.

Progress: Pericardial and liver aspiration were not attempted. Signs of pericardial involvement and liver tenderness resolved after one week and the temperature settled in the 3rd week. An E.C.G. in the 2nd week showed generalised T wave inversion. In the 3rd week the patient was transferred to a tuberculosis hospital where he remained for 6 weeks.

Four months after the first admission the patient was readmitted with a week's history of epigastric pain. Clinical examination showed an ill, pyrexial patient with signs of a pericardial effusion and a bulging mass in the epigastrium. A total of 1500 ml. of anchovy sauce pus was aspirated from the epigastric swelling and second courses of emetine and chloroquine were given. There was a rapid response to treatment, symptoms and signs cleared by the 8th day and the patient was discharged at the end of the 2nd week.

Seven months after the first admission the patient was readmitted for the 3rd time with recurrence of the original symptoms. Clinically an ill, pyrexial patient presented with signs of a pericardial effusion and a visible and palpable epigastric mass. The Hb was 10.4 g.% and the W.B.C. 12,000 per c.mm. Chest radiographs showed moderate cardiac enlargement

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with loss of normal contour and a small pleural effusion at the left base. An E.C.G. showed changes consistent with pericarditis. A 3rd course of emetine and chloroquine was started and 660 ml. of sterile anchovy sauce pus aspirated from the epigastric mass. There appeared to be a response to treatment but serial chest plates showed no decrease in the heart size. A swinging pyrexia developed in the 7th week and a further 720 ml. of pus was aspirated from the epigastric mass.

Because of the chronic relapsing nature of the liver abscess a further course of emetine and chloroquine was commenced and a laparotomy performed in the 8th week. One litre of anchovy sauce pus was removed from a large left lobe liver abscess lying immediately below the left diaphragm. No direct communication between the abscess and the pericardial cavity was found at laparotomy. Within 2 weeks of surgery the temperature settled, the liver was no longer palpable and cardiac signs resolved completely. Further radiographs showed the heart size and contour to be within normal limits, persisting elevation of the left diaphragm and a small effusion at the left base. An E.C.G. at discharge showed persisting T wave inversion. The Hb was 15.3 g.%, the W.B.C. 9,000 per c.mm. and the E.S.R. 29 mm. in one hour. The patient was discharged free of symptoms and signs in the 12th week, his relapsing illness having lasted 10 months from the first admission.

Comments: The clinical findings were interpreted as being those of a presuppurative pericardial effusion resulting from involvement of the pericardium by the inflammatory reaction at the periphery of a left lobe liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pericardial effusion.

Case Report No. 54

Patient S.M. African male Age 35 yrs. Occupation labourer

History: Continuous dull epigastric and left upper quadrant pain with radiation to the left supraclavicular region for 7 weeks. Associated symptoms were, a non-productive cough, fever and sweating. No past history of dysentery.

Examination: Temperature 99°F. Not distressed.
C.V.S.: Pulse rate 124 per minute with a good volume. B.P. 135/95 mm of Hg. The jugular venous pressure was not raised. The apex beat was within normal limits and there was a well heard 3rd heart sound.
Abdomen: Tenderness and guarding in the left upper quadrant and epigastrium and a tender liver edge palpated 2 fingerbreadths below the right costal margin.
Chest: There were no respiratory signs.

Investigations: Hb 11.8 g.%. W.B.C. 10,000 per c.mm. E.S.R.: 50 mm. in one hour.
Chest Radiographs: Slight elevation of the left diaphragm with a minimal adjacent shadowing at the left base suggestive of atelectasis. The heart size was within normal limits.
E.C.G.: Showed inverted T waves in Std. leads II & III, aVF, and chest leads V3 - V6.
Chest Screening: Normal cardiac pulsation. Decreased movement left diaphragm.
Liver Aspirate: E.histolytica present, bacteriologically sterile.
Stool: E.histolytica not seen.

Treatment: Emetine, chloroquine and diodoquin from the first day.

Progress: The left lobe of the liver was aspirated on the 2nd and 3rd days and a total of 330 ml. of anchovy sauce pus removed.

On the 2nd day, with no worsening of the patient's general condition, the neck veins were seen to be elevated to 4 cm. and a pericardial friction rub was heard intermittently during the following week. By the 10th day the signs of pericarditis and those of the liver abscess were no longer present.

Serial radiographs showed no alteration in heart size or contour with return of the left diaphragm to normal position. Further E.C.G.'s showed the inverted T waves in the chest leads to have become upright.

The patient was discharged in the 6th week having made a complete recovery. The Hb was 14.1 g.% and the W.B.C. 5,000 per c.mm. He attended follow-up after 2 weeks and one month where he was seen to have maintained clinical and radiological progress.

Comments: The clinical findings were interpreted as being those of a presuppurative pericarditis resulting from involvement of the pericardium by the inflammatory reaction at the periphery of a left lobe liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pericardial effusion.

Case Report No. 55

Patient P.M. African male Age 29 yrs. Occupation labourer

History: Continuous epigastric and retrosternal pain with radiation to the left shoulder tip for 3 weeks. Associated symptoms were, effort dyspnoea, orthopnoea, a dry cough and palpitations. No past history of dysentery.

Examination: Temperature 100°F. Ill-looking. Dyspnoeic.
C.V.S.: Pulse rate of 132 per minute with a small pulse volume and pulsus paradoxus. On inspiration B.P. 90/65 mm Hg. On expiration B.P. 100/70 mm Hg. Jugular venous pressure elevated to 4 cm. Apex beat not localised. Cardiac dullness extended beyond the normal limits to the left and on auscultation a gallop rhythm and pericardial friction rub were heard.
Chest: Diminished air entry and dullness at the left base.
Abdomen: Tenderness and guarding maximal in the epigastrium and a tender liver edge palpated 4 fingerbreadths below the costal margin.

Investigations: Hb 12 g.%. W.B.C. 15,000 per c.mm. E.S.R. 48 mm. in one hour.
Chest Radiographs: Enlargement of the cardiac shadow, with a globular outline and a cardiothoracic ratio of approximately 0.7. There was elevation of the left diaphragm with a small pleural effusion at the left base.
Chest Screening: Decreased cardiac pulsation. Decreased movement left diaphragm.
E.C.G.: S-T elevation in Std. lead I and aVL and S-T depression in chest leads V3-V4. There was flattening of the T waves in the Std. leads and the unipolar limb leads.
Liver Aspirate: E.histolytica not detected. Sterile on culture for pyogenic bacteria.
Pericardial aspirate: Serosanguineous fluid with polymorphonuclear cells predominating. E.histolytica not detected. Sterile on culture for bacteria and acid-fast bacilli. Protein 4.7 g.%.
Stool: E.histolytica not seen.

Treatment: Emetine, chloroquine and diodoquin from the first day and penicillin and streptomycin.

Progress: A left lobe liver abscess was aspirated on the 1st, 2nd and 4th days and a total of 800 ml. of thick, yellow pus removed. The 2nd aspirate was offensive and gram-negative organisms were seen on direct examination but not cultured. Antibiotics were added to the treatment and solutions of penicillin and streptomycin introduced into the abscess cavity. Pericardial aspiration on the 1st day yielded 100 ml. of uniformly blood-stained serosanguineous fluid which on examination yielded the results recorded above.

At the end of the 2nd week the jugular venous pressure was no longer raised, there was no pulsus paradoxus and the liver had decreased to 2 fingerbreadths below the costal margin. A low grade temperature settled in the 5th week.

Serial radiographs showed that the heart size had returned to normal limits after 3 weeks with clearing of the left effusion. Chest screening at this stage showed normal cardiac pulsation and diaphragmatic movement. Further E.C.G.'s showed that the S-T elevation had returned to the iso-electric line but T wave inversion persisted in the Std. leads and chest leads V_4 - V6.

The patient was free of symptoms and signs at discharge in the 9th week. There had been a weight increase of 12 pounds and the liver edge remained 2 fingerbreadths enlarged. The Hb was 12 g.% and the white cell count 9,000 per c.mm.

At follow-up after one month the patient remained well. There were no residual signs and chest radiographs and the E.C.G. were normal.

Comments: The clinical findings were interpreted as being those of a presuppurative pericardial effusion resulting from involvement of the pericardium by the inflammatory reaction at the periphery of a left lobe liver abscess.

Diagnosis: Amoebic liver abscess with a presuppurative pericardial effusion.

Case Report No. 56

Patient A.N. African male Age 24 yrs. Occupation labourer

History: Low retrosternal and epigastric pain for 4 weeks with radiation to the left shoulder tip. Associated symptoms were a non-productive cough, dyspnoea and giddiness. No past history of dysentery.

Examination: Temperature 100°F. Not distressed. Pale mucosae. Sacral oedema.
C.V.S.: Pulse rate 90 per minute, small pulse volume and pulsus paradoxus. Jugular venous pressure raised to the angle of the jaw. B.P. 90/65 mm Hg. Cardiac dullness increased to the left. Apex beat not palpable. On auscultation there was a gallop rhythm and distant heart sounds.
Chest: Diminished movement, dullness on percussion, decreased air entry and intercostal tenderness at the right base. Localised intercostal tenderness at the left base.
Abdomen: Tenderness and guarding maximal in the epigastrium, also detected in the right upper quadrant. Liver edge palpated 2 finger-breadths below the costal margin.

Investigations: Hb 9.8 g.%. W.B.C. 9,000 per c.mm. E.S.R. 62 mm. in one hour.
Chest Radiegraphs: Increase in the transverse diameter of the heart, cardiothoracic ratio of 0.65, and a left ventricular configuration. Minimal elevation of the right diaphragm and a pleural reaction at the base of the right lung.
Chest Screening: Reduced cardiac pulsation and adequate movement of both hemidiaphragms.
E.C.G.: S-T segment elevation in Std. leads 1 and 2 and avL. Inverted T waves in Std. leads 3 and AvF., biphasic T waves in V4 and flat T waves in V5 and V6.
Liver Aspiration: Anchovy sauce pus obtained. E.histolytica not identified or cultured. Sterile on culture for pyogenic bacteria.
Pericardial Aspiration: Anchovy sauce pus obtained. E.histolytica not seen or cultured. Sterile on culture for pyogenic bacteria and A.F.B.
Stool: Trophozoites of E.histolytica.
Progress: The day after admission exploratory aspiration of the left lobe of the liver yielded 70 ml. of thick anchovy sauce pus. Pericardial aspiration via the epigastric route yielded 85 ml. of thick anchovy sauce pus.

The temperature settled on the 6th day and the patient was symptom free after one week. A pericardial friction rub became audible on the 7th day. The jugular venous pressure settled on the 14th day but the pericardial friction rub and gallop rhythm persisted until the 20th day.

At discharge on the 31st day there were no residual symptoms and signs and there had been a weight increase in hospital of 3 pounds. The Hb was 12 g.%, the W.B.C. 7,000 per c.mm. and the E.S.R. 34 mm. in one hour. Chest radiegraphs showed the heart size to be within normal limits and clearing of the changes at the base of the right lung. An

E.C.G. showed T wave inversion in all leads. Six months after discharge clinical and radiological progress had been maintained and the E.C.G. was normal.

Comments: The clinical findings were interpreted as being those of suppurative amoebic pericarditis which had resulted from extension of a left lobe liver abscess into the pericardium. There was also clinical evidence of an abscess in the right lobe of the liver.

Diagnosis: Amoebic liver abscesses with suppurative amoebic pericarditis.

Case Report No. 57

Patient A.N. African male Age 35 yrs. Occupation labourer

History: Epigastric and retrosternal pain for 3 months associated with increasing dyspnoea on exertion and a non-productive cough. Three days before admission there was a sudden increase in the severity of pain and dyspnoea. No past history of dysentery.

Examination: Temperature subnormal. Ill-looking. Dyspnoeic. Wasted. Pale mucosae. Peripheral vascular collapse.
C.V.S.: Jugular venous pressure raised. Pulse rate 120 per minute, poor pulse volume. B.P. 80/40 mm Hg. The apex beat was not localised but diffuse precordial pulsation visualised and palpated. A gallop rhythm was heard on auscultation.
Chest: Respiratory rate 38 per minute, otherwise normal.
Abdomen: Tender 4 fingerbreadths enlarged liver.

Investigations: Hb 11.2 g.%. W.B.C. 12,000 per c.mm. E.S.R. 50 mm. in one hour.
Chest Radiographs: Marked increase in the transverse diameter of the heart with a bulge along the left cardiac border which resembled a cardiac aneurysm. The lung fields were clear and the diaphragms in normal position.
E.C.G.: Rsr' pattern in lead I and aVL with S-T segment elevation in these leads. A QrS pattern with depressed S-T segments and inverted T waves was present in lead II, III, and aVF. T wave inversion was noted throughout the chest leads.
Stool: E.histolytica not seen.

Treatment: Digitalis, diuretics and penicillin were started on admission. Chloroquine was added to the above regime on the 3rd day.

Progress: The patient's condition deteriorated during the first 3 days and there was no response to the above treatment. Diagnoses considered were, cardiac aneurysm, aortic aneurysm, pericardial effusion, cardiac failure of undetermined etiology and a left lobe amoebic liver abscess. The patient died on the 4th day.

Necropsy Findings: The pericardial sac contained approximately 400 ml. of blood-stained pus. The visceral and parietal pericardium were grossly thickened and covered with a shaggy exudate. The superior surface of the left lobe of the liver and the left diaphragm were adherent. There was a communication approximately 2 cm. in diameter between the pericardial sac and a left lobe liver abscess. Ulcers resembling amoebic ulcers were found in the caecum.

Histology revealed an organising fibrinous pericarditis. The myocardium was normal. Liver sections showed histological changes consistent with a chronic liver abscess, siderosis and increased portal

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fibrosis. *E.histolytica* were found in the walls of the liver abscess and in the margins of the caecal ulcers.

Comments: Suppurative amoebic pericarditis resulting from extension of a left lobe liver abscess was not seriously considered as an explanation of the clinical findings and the diagnosis was only established at necropsy.

Diagnosis: Amoebic colitis and amoebic liver abscess with suppurative amoebic pericarditis.

Case Report No. 58

Patient N.N. African male Age 49 yrs. Occupation labourer

History: Continuous epigastric pain for 4 months. Associated symptoms were a non productive cough, anorexia and weight loss. No past history of dysentery.

Examination: Temperature subnormal. Evidence of recent weight loss.
Chest: Normal.
C.V.S.: Pulse rate 120 per minute, B.P. 130/80 mm Hg, otherwise normal.
Abdomen: Epigastric guarding and tenderness. Tender 4 fingerbreadths enlarged liver. Firm non-tender enlarged spleen.

Investigations: Hb 12.4 g.%. W.B.C. 13,000 per c.mm. E.S.R. 51 mm. in one hour.
Chest Radiographs: There was an increase in the transverse diameter of the heart and a localised prominence along the left ventricular border. A diagnosis of a cardiac aneurysm was suggested by the radiologist.
Chest Screening: Normal diaphragmatic movement. The rounded shadow on the left cardiac border pulsated, and was thought by the radiologist to be an aneurysm of the left ventricle.
E.C.G.: T wave inversion in standard leads II and III, aVL, and in the chest leads V1 - V3.
Stool: E.histolytica not seen.

Treatment & Progress: On admission the clinical presentation was thought to be that of a left lobe amoebic liver abscess and the patient was treated with emetine and chloroquine. On the 3rd day signs of cardiac failure were noticed. These were associated with a systolic murmur suggestive of tricuspid incompetence and an early diastolic murmur suggestive of aortic incompetence. Emetine and chloroquine were discontinued and the patient was treated for cardiac failure and investigated and treated for subacute bacterial endocarditis. Digitalis and mersalyl with the antibiotics penicillin and streptomycin were administered.

Pyrexia persisted and the cardiac failure did not respond to treatment. The patient's condition gradually deteriorated, he developed a left-sided pleural effusion and died on the 35th day.

Necropsy Findings: Exposure and incision of the pericardium showed the cavity to contain approximately 800 ml. of anchovy sauce pus. There was inflammatory thickening of the visceral and parietal layers and a shaggy exudate was seen on their opposing surfaces. An opening approximately 1 cm. in diameter connected the pericardial cavity through the left diaphragm with a posteriorly situated left lobe liver abscess which was approximately 8 cm. in diameter. A second opening was found in the lower left posterior aspect of the parietal pericardium connecting the pericardial sac with the left pleural cavity and posterior mediastinum.

The heart valves were normal and there was no evidence of subacute bacterial endocarditis. Seven hundred ml. of anchovy sauce pus was found in the left pleural cavity and there was inflammatory thickening of both pleural layers. Loculated pockets of pus were found in the posterior mediastinum. Ulcers consistent with amoebic ulcers were seen in the caecum. The left cerebellar hemisphere contained a small focus of cerebral softening.

Histological examination revealed amoeba in the walls of the liver abscess, in sections of the pericardium, in the margins of the caecal ulcers and in the substance of the brain abscess.

Comments: The original correct diagnosis of a left lobe amoebic liver abscess was unfortunately discarded in favour of a diagnosis of cardiac failure with subacute bacterial endocarditis. No explanation was found for the cardiac murmurs at necropsy.

Diagnosis: Amoebic colitis, amoebic liver abscess, suppurative amoebic pericarditis, amoebic empyema and an amoebic brain abscess.

Case Report No. 59

Patient A.M. African male Age 48 yrs. Occupation labourer

History: Continuous epigastric and lower substernal pain for 5 weeks. Associated symptoms were, increasing effort dyspnoea, a non-productive cough and fatigue. Past history of dysentery 2 years ago.

Examination: Subnormal temperature. Ill-looking. Emaciated. Cold extremities.
C.V.S.: Pulse rate 132 per minute, small pulse volume and marked pulsus paradoxus. B.P. 100/70 mm Hg. Jugular venous pressure raised to 4 cm. The apex beat was not palpable, and cardiac dullness extended to the left anterior axillary line. The heart sounds were muffled and there was a gallop rhythm.
Chest: Respiratory rate 36 per minute. Bilateral basal crepitations.
Abdomen: Tenderness and guarding maximal in the epigastrium and a 4 fingerbreadths tender enlarged liver.

Investigations: Hb 11 g.%. W.B.C. 5,000 per c.mm. E.S.R. 57 mm. in one hour.
Chest Radiographs: These showed massive cardiac enlargement with a globular cardiac outline. The diaphragms were in normal position and the visible lung fields were not congested.
Chest Screening: Absent cardiac pulsation. Immobility of left diaphragm.
Pericardial aspiration: E.histolytica present. Sterile on culture for pyogenic bacteria and acid-fast bacilli. Liver cells detected in the pericardial aspirate.
E.C.G.: Serial E.C.G.'s showed low voltage in all leads with T wave inversion throughout. There was no significant change in the above pattern during hospitalisation.
Stool: E.histolytica not seen.

Treatment: Emetine, chloroquine and diodoquin from the first day. Penicillin and streptomycin were added to the above regime.

Progress: On admission 600 ml. of anchovy sauce pus was aspirated from the pericardium through the left upper epigastrium. Liver cells and Entamoeba histolytica were found in the aspirate. The pericardial cavity was aspirated 10 times in the first 3 weeks and a total of 6 litres of pus removed. Escherichesa coli were cultured from 2 of the aspirates, the fluid gradually changed to a dirty yellow colour and contained increasing quantities of thick debris. Tetracycline followed by chloramphenicol was added to the treatment.

Sixty ml. of air was introduced into the pericardial cavity and the air-fluid level formed showed the parietal pericardial layer to be approximately one centimeter in thickness. Twenty ml. of contrast media injected into the pericardium demonstrated a posterior communication with the left subphrenic region.

On the 23rd day a laparotomy was performed and adhesions were demonstrated between the superior surface of the left lobe of the liver and the left hemidiaphragm. At laparotomy a liver abscess was not visualised or localised by needle aspiration and tube drainage of the pericardium was instituted through the diaphragm. This drained a further 1300 ml. of pus in the 4th week and thereafter drainage ceased.

Clinically during the first month signs of a pericardial effusion with a tendency to tamponade persisted. On 3 occasions signs of severe tamponade with peripheral vascular collapse necessitated emergency aspiration of the pericardium. Quantities of up to 1200 ml. of pus were aspirated resulting in dramatic clinical improvement.

Persistence of a raised jugular venous pressure, pulsus paradoxus, a small pulse pressure, a third heart sound and hepatomegaly during the 2nd month suggested the onset of constrictive pericarditis. The decrease in the transverse diameter of the heart seen on serial radiographs supported this clinical impression. Chest screening at this stage showed the heart size to be within normal limits, no visible cardiac pulsation and an immobile left diaphragm.

At the end of the 2nd month signs and symptoms of constrictive pericarditis gradually increased in severity. A right-sided pleural effusion developed from which a litre of sterile serous fluid was aspirated. In consultation with the thoracic surgeons it was decided to attempt a pericardectomy. At thoracotomy a small non-pulsatile heart was found. There was inflammatory thickening of the pericardial layers and excision of the visceral layer resulted in dramatic freeing of the underlying myocardium which bulged prominently through the incision. Difficulty was experienced in stripping the pericardial tissue from the ventricles. Post-operatively the blood pressure fell, there was continued deterioration with death on the 2nd post-operative day.

Necropsy Findings: The heart size was within normal limits and there was marked inflammatory thickening of the parietal and visceral pericardium. The visceral layer was approximately 5 mm. in thickness and a small quantity of shaggy exudate was found on the opposing surfaces of the pericardial membranes. It was impossible to strip the visceral pericardium from the myocardium without tearing away considerable portions of muscle. A communication was found between the pericardial cavity and the subphrenic space. This opening measured approximately 1 cm. in diameter.

The liver was enlarged 3 fingerbreadths below the right costal margin and its cut surface showed a typical nutmeg appearance. A small focus of scar tissue thought to represent the remains of a healed liver abscess was found in the left lobe immediately below the opening in the left diaphragm.

Small pleural effusions were found in both pleural spaces and there was consolidation of the lower lobe of the left lung. Examination of the kidneys showed multiple small focal abscesses.

Histology: Sections of the pericardium consisted predominantly of fibrous tissue but scattered foci of granulation tissue and evidence of fibroblastic proliferation were seen. There was round cell infiltration in the superficial layers of the myocardium. No amoeba were seen in the above sections. Liver sections revealed centrilobular congestion and necrosis. The histology of the left lower lobe of the lung was consistent with lobar pneumonia. Sections of the kidneys showed the picture of suppurative pyelonephritis.

Comments: Suppurative amoebic pericarditis resulted from extension of a left lobe liver abscess into the pericardium. The pericardium subsequently became secondarily infected with Escherichese coli. The clinical impression of a rapidly progressing constrictive pericarditis was confirmed at thoracotomy and necropsy.

Diagnosis: Amoebic liver abscess with suppurative amoebic pericarditis progressing to constrictive pericarditis.

Case Report No. 60

Patient M.M. African male Age 40 yrs. Occupation labourer

History: Continuous retrosternal chest pain aggravated by effort and coughing for 6 weeks. Associated symptoms were epigastric discomfort, a non-productive cough, dyspnoea on exertion, swelling of the feet and weight loss. Two years before admission twenty-five ounces of amoebic pus was aspirated from a right lobe liver abscess at this hospital.

Examination: Temperature subnormal. Ill-looking. Evidence of recent weight loss. Pale mucosae. Ankle and sacral oedema.
C.V.S.: Pulse rate 96 per minute, small pulse volume with marked pulsus paradoxus. Jugular venous pressure raised to the angle of the jaw. Blood pressure on inspiration of 100/90 mm Hg and on expiration 110/95 mm Hg. The apex beat was not palpated and cardiac dullness increased to the left and percussed to the right of the sternum. The heart sounds were muffled, there was an audible gallop rhythm and pericardial friction rub.
Chest: Tachypnoea with a stony dull percussion note and absent air entry at the right base.
Abdomen: A 4 fingerbreadths tender enlarged liver palpated with a localised swelling and maximal tenderness in the epigastrium.

Investigations: Hb 10.4 g.%. W.B.C. 22,000 per c.mm. E.S.R. 54 mm. in one hour.
Chest Radiographs: The heart was globular in shape and the cardiothoracic ratio 0.75. The lung fields were clear and there was a small right-sided pleural effusion.
Chest Screening: Absent cardiac pulsation. Decreased movement of the left diaphragm.
E.C.G.: Low voltage in the standard and augmented unipolar limb leads with flattened or inverted T waves in all leads.
Liver Aspirate: E.histolytica present. Light growth of Staphylococcus pyogenes obtained on culture.
Stool: E.histolytica not seen.

Treatment: Emetine, chloroquine and diodoquin from the 1st day and penicillin and streptomycin. The patient was digitalised and intramuscular mersalyl given twice weekly.

Progress: On admission 510 ml. of anchovy sauce pus was aspirated from the left upper epigastrium. During the first 10 days 4 epigastric aspirations yielded a total of 1020 ml. of pus. On the 10th day direct pericardial aspiration was attempted through the left fourth interspace and only a few ml. of pus obtained. Following the final epigastric aspiration the patient developed a left-sided pleural effusion from which 690 ml. of serosanguineous fluid was removed.

Clinically the signs of pericardial effusion found on admission persisted for the first 2 months. In the 3rd month the above signs worsened and dyspnoea increased. Serial radiographs of the chest showed a progressive decrease in the transverse diameter of the heart which was well within normal limits at 4 weeks. Chest screening in the 6th and 10th weeks showed a small heart with minimal pulsation of the left border, no pulsation of the right border and normal movement of the diaphragms. Further E.C.G.'s showed a low voltage pattern to have developed with flattening of the T waves throughout.

As a result of the above features of constrictive pericarditis a pericardectomy was attempted on the 73rd day. At thoracotomy the thickened pericardial layers over the left ventricle did not appear to be interfering greatly with contraction. There was a thick band of pericardial tissue lying across and obstructing the pulmonary outflow tract. This was dissected and stripped with a striking increase in right ventricular and pulmonary artery pulsation. During the 2 weeks following pericardectomy there was a noticeable improvement in the clinical condition. The pulse pressure increased, the jugular venous pressure fell and the liver size decreased to 2 fingerbreadths below the costal margin.

At discharge in the 24th week there was no clinical evidence of heart disease. In spite of loss of oedema fluid there had been a weight increase of 17 lbs. during hospitalisation. His effort tolerance was normal. E.C.G. showed a general increase in voltage with a return to normal position of the T waves. The Hb was 15 g.%, the W.B.C. 11,000 per c.mm. and the E.S.R. 27 mm. in one hour.

At follow-up at 6 and 12 months the above progress had been maintained and chest radiographs and E.C.G.'s showed no residual changes.

Comments: The clinical findings were interpreted as being those of a suppurative amoebic pericarditis following extension of a left lobe liver abscess into the pericardium. The clinical impression of progress to a stage of constrictive pericarditis was confirmed at thoracotomy.

Diagnosis: Amoebic liver abscess with suppurative amoebic pericarditis progressing to constrictive pericarditis.

Case Report No. 61

Patient B.D. African male Age 23 yrs. Occupation labourer

History: Pain in the lower anterior chest and precordial region for 2 months. Associated symptoms were a dry cough, dyspnoea on exertion and swelling of the feet. No past history of dysentery.

Examination: Temperature 99°F. Not distressed. Finger clubbing. Ankle oedema.
C.V.S.: Jugular venous pressure raised to the angle of the jaw. Pulse rate 124 per minute, with pulsus paradoxus. B.P. of 105/65 mm Hg. The apex beat was not palpable. On percussion the area of cardiac dullness was increased to the left. The heart sounds were muffled and there was an audible third sound.
Chest: Dullness and diminished air entry at the left base.
Abdomen: Tenderness and guarding maximal in the epigastrium and a 3 fingerbreadth enlarged tender liver.

Investigations: Hb 9.6 g.%. W.B.C. 16,000 per c.mm. E.S.R. 58 mm. in one hour.
Chest Radiographs: Massive cardiomegaly with a globular outline to the heart and a cardiothoracic ratio greater than 0.7. The visible lung fields were clear.
Chest Screening: Absent cardiac pulsation. Immobile left diaphragm.
E.C.G.: T wave inversion in all leads except aVR and V1.
Pericardial aspiration: Trophozoites of E.histolytica present.
Sterile on culture for pyogenic organisms and acid-fast bacilli.
Stool: E.histolytica not seen.

Treatment: Penicillin and streptomycin had been given for one week before hospitalisation. Emetine, chloroquine and diodoquin were given from the first day. The patient was digitalised and intramuscular mersalyl injected twice weekly.

Progress: On admission 1500 ml. of anchovy sauce pus was aspirated from the pericardium through the epigastrium. There was an immediate noticeable fall in the neck veins, the blood pressure rose to 120/70 mm Hg, the pulsus paradoxus was less noticeable and a pericardial friction rub was heard. Four pericardial aspirations were done during the first ten days bringing the total aspirate to 2,150 ml. of pus which remained sterile on culture for acid-fast bacilli and bacteria.

Sixty ml. of air was introduced into the pericardial cavity and radiographs showed the parietal pericardial layers to be about one centimeter in thickness. Serial radiographs showed gradual absorption of the air with the heart size returning to within normal limits after one month. Chest screening in the 8th week showed the heart size and contour to be normal with satisfactory cardiac pulsation and a fixed left diaphragm. Radiographs at discharge in the 8th week were normal except for minimal residual pleural thickening at the left base.

The temperature settled on the 6th day but clinical progress was unimpressive in the first month. Signs of cardiac embarrassment such as the raised neck veins, pulsus paradoxus and the large liver persisted. The patient developed a tachycardia of 120 to 130 per minute which lasted 2 weeks and the blood pressure fell to 85/65 mm Hg. With the decrease in heart size radiologically the onset of constrictive pericarditis seemed likely. Remarkable improvement occurred in the 5th week. The venous pressure fell, oedema cleared, the B.P. rose to 120/80 mm Hg, pulsus paradoxus disappeared and the liver edge decreased to one fingerbreadth below the costal margin.

At discharge in the 8th week the patient appeared to have recovered completely. There was dullness on percussion at the left base and the jugular venous pressure persisted at 4 cm. above the sternum. An E.C.G. showed universal T wave inversion. Whilst in hospital there had been a weight increase of 19 pounds. The Hb had risen to 12.4 g.%, the W.B.C. was 9,000 per c.mm. and the E.S.R. 18 mm. in one hour.

At follow-up after 6 months, progress was maintained, there was no clinical evidence of constriction, a chest radiograph and E.C.G. were normal.

Comments: The clinical findings were interpreted as being those of a suppurative amoebic pericarditis following extension of a left lobe liver abscess through the diaphragm. Following pericardial drainage the patient appeared to be developing constrictive pericarditis but subsequently made a complete recovery.

Diagnosis: Amoebic liver abscess with suppurative amoebic pericarditis.

Case Report No. 62

Patient J.M. African male Age 30 years Occupation labourer

History: Low retrosternal pain, pleuritic pain in the right lower chest and cough for one year. Three days prior to admission sudden increase in the retrosternal pain with dyspnoea. No past history of dysentery.

Examination: Temperature 100°F. Ill-looking.
C.V.S.: Pulse rate 96 per minute, no pulsus paradoxus. B.P. 110/70 mm.Hg. Jugular venous pressure raised to the angle of the jaw. Apex beat not palpable. Area of cardiac dullness increased to the left. There was a pericardial friction rub and an audible third heart sound.
Chest: Respiratory rate 32 per minute, otherwise normal.
Abdomen: Epigastric guarding and tenderness with a 2 fingerbreadths tender enlarged liver.

Investigations: Hb 11 g.%. W.B.C. 16,000 per c.mm. E.S.R. 43 mm. in one hour.
Chest Radiographs: Increase in the transverse diameter of the heart. Moderate elevation of the right diaphragm, patchy consolidation and a small effusion at the right base.
Chest Screening: Reduced cardiac pulsation. Immobile left and right diaphragm.
E.C.G.: No changes suggestive of pericardial involvement.
Pericardial Aspirate: E.histolytica not detected.
Stool: E.histolytica not seen.

Treatment: Streptomycin, isoniazid and pyridoxine were started on the 2nd day. Emetine, chloroquine and diodoquin from the 6th day.

Progress: On the 6th day there was a sudden increase in the retrosternal pain and dyspnoea and the patient complained of epigastric pain. Examination revealed peripheral vascular collapse, a pulse rate of 132 per minute, a small volumed pulse and pulsus paradoxus. The B.P. was 90/70 mm. Hg. Radiographs showed further increase in the transverse diameter of the heart. Pericardial aspiration yielded 320 ml. of thin anchovy sauce pus. No amoeba, bacteria or acid-fast bacilli were seen on direct examination or cultured from the pus.

Following aspiration the pulse rate fell, pulsus paradoxus was less obvious, there was a noticeable decrease in the height of the neck veins and the B.P. rose to 130/75 mm. Hg. Emetine, chloroquine and diodoquin were commenced at this stage. Recurrence of the cardiac tamponade was relieved by a second aspiration on the 8th day which yielded 465 ml. of anchovy sauce pus. Further attempts at pericardial aspiration failed.

During the first 5 weeks clinical signs suggestive of a rapidly developing constrictive pericarditis developed. The jugular venous pressure remained elevated, there was a gradual fall in the

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blood pressure, a decrease in the pulse pressure and marked pulsus paradoxus of more than 10 mm. of Hg. Chest screening in the 5th week showed a normal sized heart with absence of cardiac pulsation. E.C.G. at this stage showed a low voltage pattern with inverted T waves throughout.

Because of what appeared to be a rapidly developing constrictive pericarditis a thoracotomy was performed on the 44th day. The right pleural cavity was seen to contain a serosanguineous effusion. The pericardial layers consisted of thickened inflammatory tissue with shaggy exudate on their opposing surfaces. The presence of a constrictive pericarditis was verified when on incising the visceral pericardium there was a dramatic bulging of the left ventricle through the incision. The visceral layer was stripped from the ventricles without difficulty. Following the removal of the visceral layer there was an impressive increase in cardiac pulsation. The procedure was concluded but one hour later when the patient's condition appeared satisfactory there was an unexplained cardiac arrest. A cardiac massage was attempted but there was no response.

Necropsy Findings: There was evidence of a recent thoracotomy. The pleural layers related to the heart were thickened and adherent. Most of the pericardial layer surrounding the right and left ventricles had been removed. The residual parietal and visceral pericardia were thickened with shaggy exudate on their opposing surfaces. The coronary arteries were normal. The right pleural cavity contained a serosanguineous effusion.

The left diaphragm was thickened and adherent to the left lobe of the liver. A chronic abscess approximately 5 cm. in diameter was found in the left lobe of the liver immediately below the left diaphragm. There was a communication a few millimeters in diameter between the liver abscess and the pericardial cavity. A focus of scar tissue was found in the right lobe of the liver.

At histology no amoebae were seen in sections of the liver abscess or pericardium. Sections of the visceral pericardium consisted predominantly of fibrous tissue with areas of non-specific inflammation.

Comments: Constrictive pericarditis developed with surprising rapidity in this patient.

Diagnosis: Amoebic liver abscess with suppurative amoebic pericarditis progressing to constrictive pericarditis.

Case Report No. 63

Patient J.N. African male Age 21 yrs. Occupation labourer

History: Continuous low retrosternal pain aggravated by respiration and dyspnoea on exertion for 3 months. The patient was treated for an amoebic liver abscess of the right lobe of the liver 5 months ago. No past history of dysentery.

Examination: Temperature 98°F. Ill-looking. Pale mucosa.
C.V.S.: Jugular venous pressure raised to angle of jaw. Pulse rate 130 per minute, small pulse volume with pulsus paradoxus. B.P. of 90/60 mm. Hg. Apex beat not localised. Cardiac dullness increased to left of the mid-clavicular line. On auscultation there was a gallop rhythm with a pericardial friction rub.
Chest: Dullness on percussion, decreased air entry with a pleural rub at the right base.
Abdomen: 4 fingerbreadths enlarged tender liver with area of maximum tenderness in the epigastrium.

Investigations: Hb 8.2 g.%. W.B.C. 25,000 per c.mm. E.S.R. 61 mm. in one hour.
Chest Radiographs: Increase in the transverse diameter of the heart with a globular cardiac outline. Lung fields clear and the diaphragms in normal position.
Chest Screening: Absent cardiac pulsation. Immobile right diaphragm.
E.C.G.: Flattened T waves in Std. lead I, aVL, with T wave inversion in remaining leads.
Pericardial Aspirate: 480 ml. anchovy sauce pus. No amoeba detected.
Liver Aspirate: E.histolytica not detected.
Stool: E.histolytica not detected.

Treatment: Emetine, diodoquin and chloroquine from the 1st day. Tetracycline 250 mg. q.i.d. x 10 days.

Progress: One hundred ml. of thick anchovy sauce pus was aspirated from a left lobe liver abscess. A total of 740 ml. of anchovy sauce pus was aspirated from the pericardium in the first 2 days. Air introduced at the second aspiration produced an air-fluid level which radiologically revealed minimal thickening of the parietal pericardium.

The temperature settled on the 12th day and signs of a pericardial effusion cleared in the 4th week with a rise in the B.P. to 110/70 mm Hg. Chest screening in the 5th week showed a normal heart size with normal cardiac pulsation and paresis of the right hemidiaphragm.

At discharge on the 44th day there were no residual symptoms or signs; radiographs showed the heart size and contour to be within normal limits. The Hb had risen to 13.9 g.%, the W.B.C. was 7,000 per c.mm. and the E.S.R. 25 mm. in one hour.

At follow up after 3 months there were no symptoms or signs. Chest radiographs and the E.C.G. were normal.

Comments: The clinical findings were interpreted as being those of suppurative amoebic pericarditis following extension of a left lobe liver abscess into the pericardium. There was radiological evidence of an associated abscess in the right lobe of the liver.

Diagnosis: Amoebic liver abscesses with suppurative amoebic pericarditis.

Case Report No. 64

Patient J.D. African male Age 39 yrs. Occupation labourer

History: Continuous pain over the left chest and precordial region for one month. The pain radiated to the left shoulder. Associated symptoms were fever, weight loss and swelling of the feet for one month.

Twenty-four hours before admission there was a sudden exacerbation of the chest pain with collapse and severe dyspnoea. No past history of dysentery.

Examination: Temperature 101°F. Distressed. Dyspnoeic. Ankle oedema. Cold extremities. Pale mucosae.
C.V.S.: Pulse rate of 130 per minute with a small pulse volume and pulsus paradoxus. B.P. of 95/65 mm Hg. Jugular venous pressure raised to the angle of the jaw. Cardiac dullness was increased to the left and the apex beat was not palpable. The heart sounds were muffled, there was a gallop rhythm and a pericardial friction rub.
Chest: Bronchial breathing and coarse crepitations at the left base.
Abdomen: Tenderness and guarding maximal in the epigastrium and a tender liver edge palpated 2 fingerbreadths below the costal margin.

Investigations: Hb 8.4 g.%. W.B.C. 12,000 per c.mm. E.S.R. 58 mm. in one hour.
Chest Radiographs: The transverse diameter of the heart was greatly increased, the cardiothoracic ratio was 0.7 and the cardiac outline globular in shape. Elevation of the left diaphragm was present with shadowing in the adjacent left lower lung field suggestive of atelectasis.
Chest Screening: Absent cardiac pulsation. Immobile left diaphragm.
E.C.G.: S-T segment elevation in Std. leads I & II and aVL. S-T segment depression and T wave inversion in Std. lead III and aVF.
Liver Aspirate: E.histolytica not detected. Sterile on culture for pyogenic bacteria.
Pericardial Aspirate: E.histolytica present. Sterile on culture for pyogenic organisms and acid-fast bacilli.
Stool: E.histolytica not seen.

Treatment: Emetine, chloroquine and diedoquin from the first day. Penicillin and tetracycline were added to the above treatment. The patient was digitalised and intramuscular mersalyl given twice weekly.

Progress: On admission 500 ml. of thin anchovy sauce pus was aspirated from the pericardium through the epigastrium and 200 ml. of thick anchovy sauce pus removed from an abscess in the left lobe of the liver. The pericardial aspiration resulted in a slowing of the pulse, a rise in the B.P. to 120/75 mm.Hg and noticeable symptomatic relief. During the first 4 days 4 pericardial aspirations were carried out and a total of 920 ml. of anchovy sauce pus aspirated. E.histolytica was isolated from the fourth aspirate and all specimens were sterile when cultured for pyogenic organisms and acid-fast bacilli.

The pyrexia settled on the 10th day. Signs of pericardial involvement such as the pulsus paradoxus, the third heart sound and the raised jugular venous pressure cleared after 2 weeks' treatment. The blood pressure remained at 80/60 mm Hg until the 4th week when it rose to 115/70 mm Hg. The apex beat was easily palpated at this stage.

Radiographs on the 4th day revealed an air-fluid level in the pericardial sac which indicated that the parietal pericardium was about ½ cm. in thickness. Serial radiographs showed a gradual decrease in heart size which was within normal limits at 4 weeks. Chest screening at this stage showed normal cardiac pulsation but restricted movement of the left diaphragm. Serial E.C.G.'s showed T wave inversion to have developed and this was followed by a return to a normal tracing in the 5th week.

The patient was discharged in the 7th week with no cardiac signs and symptoms or residual chest signs. Whilst in hospital in spite of the loss of oedema fluid there was a weight increase of 8 pounds. At discharge the Hb was 13.8 g.%, the W.B.C. 7,000 per c.mm. and the E.S.R. 7 mm. in one hour.

At follow-up after one and 6 months clinical progress had been maintained and there was no clinical radiological or electrocardiographic evidence of constrictive pericarditis.

Comments: The clinical findings were interpreted as being those of suppurative amoebic pericarditis following extension of a left lobe abscess into the pericardium.

Diagnosis: Amoebic liver abscess with suppurative amoebic pericarditis.

Case Report No. 65

Patient S.B. African male Age 22 yrs. Occupation bus conductor

History: Periodic episodes of a stabbing upper abdominal pain for 3 months. Eight days before admission the pain became continuous in nature, was situated in the epigastrium and radiated to the lower thoracic region. There were no cardiac or respiratory symptoms and there was no past history of dysentery.

Examination: Temperature 102°F. Ill-looking. Emaciated. Pale mucosa.
Abdomen: Moderate abdominal distension. Guarding and tenderness with a 3 fingerbreadths tender hepatomegaly with maximal tenderness in the epigastrium.
C.V.S.: Pulse rate 110 per minute, blood pressure 110/60 mm Hg., otherwise normal.
Chest: Dullness and diminished air entry at the base of the left lung.

Investigations: Hb 7.7 g.%. W.B.C. 34,000 per c.mm. E.S.R. 64 mm. in one hour.
Chest Radiographs: Pleural effusion at the left base. The heart size was within normal limits.
Abdominal Radiographs: Gaseous distension of large and small bowel.
Liver Aspirate: E.histolytica not detected. No bacteria cultured.
Stool: E.histolytica not detected.

Treatment: Emetine, chloroquine and diodoquin from the first day. Tetracycline was added to the treatment.

Progress: The presence of a left lobe liver abscess was established on the 2nd day when 200 ml. of greenish-yellow pus was aspirated from the left epigastrium. On the 3rd day localised intercostal tenderness was found at the right base and aspiration of anchovy sauce pus through the lower right rib spaces established the diagnosis of an associated abscess of the right lobe of the liver.

The patient collapsed on the 4th day and signs of a pericardial effusion were found. There was a raised jugular venous pressure, pulsus paradoxus, a blood pressure of 75/55 mm. Hg, muffled heart sounds and a pericardial friction rub. Chest radiographs showed an increase in the transverse diameter of the heart and a globular cardiac outline. Only 20 ml. of serous fluid was obtained at the first pericardial aspiration. Second and third aspirations yielded 100 ml. and 180 ml. of thick blood-stained pus. No amoebae, pyogenic organisms or acid-fast bacilli were cultured from the pericardial pus. Serial radiographs showed the heart size to have returned to within normal limits after 2 weeks. An E.C.G. at this time showed flattened or inverted T waves in all leads.

The clinical signs of cardiac embarrassment did not resolve.

The raised jugular venous pressure, peripheral oedema, hepatomegaly, a blood pressure of 90/65 mm. Hg, and the pulsus paradoxus persisted. In the 4th week digitalis and mersalyl were added to the treatment. Chest screening in the 10th week showed no pulsation of the cardiac contour with normal position and movement of the right diaphragm. The left diaphragm was obscured by a pleural effusion.

During the first 10 weeks there was a gradual increase of the left pleural effusion and ten aspirations yielded 4,800 ml. of serous or serosanguineous fluid. The aspirates contained polymorphs with a few lymphocytes, the protein content varied from 2 - 2.5 g.% and no pyogenic or acid-fast bacilli were cultured.

Repeated episodes of right lower chest pain with frank haemoptysis occurred from the 9th to the 12th week. Signs of right basal consolidation with a pleural rub were found and radiographs showed a zone of consolidation in the right lower lobe. The above clinical findings were thought to have been produced by multiple pulmonary emboli and anticoagulant therapy was commenced.

In consultation with the thoracic surgeons and cardiologists a pericardectomy was considered when signs of constrictive pericarditis showed no tendency to abate after 3 months. Fortunately there was a delay of 6 weeks before surgery could be done. During this period there was a remarkable improvement in the clinical picture.

The improvement in the patient's condition continued and at discharge on the 179th day there were no symptoms and there had been a weight increase during hospitalisation of 27 pounds. The jugular venous pressure remained elevated to 3 cm., the blood pressure was 115/85 mm. of Hg and there were no signs of pericardial involvement. Chest screening showed a normal sized heart and adequate cardiac pulsation. The Hb was 12.6 g.%, the W.B.C. 11,000 per c.mm. and the E.S.R. 29 mm. in one hour.

At follow-up after one month the patient remained well, the jugular venous pressure persisted at 3 cm. and an E.C.G. and chest radiographs were normal.

Comments: The clinical findings were interpreted as being those of a left lobe amoebic liver abscess which extended into the pericardium producing a suppurative amoebic pericarditis. Following treatment of the suppurative pericarditis the patient appeared to be developing constrictive pericarditis but the features suggestive of this complication resolved with further observation.

Diagnosis: Amoebic liver abscesses with suppurative amoebic pericarditis.

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