

SUBPHRENIC ABSCESS*

BY

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INTRODUCTION

Subphrenic infection occurring in the hidden borderland between thorax and abdomen is difficult to diagnose and to treat. Mistakes in management may be calamitous for the patient. The problems are enhanced because the clinical picture is often obscured by the causal condition and by previous operative intervention. The condition, moreover, is one with which few surgeons have had extensive experience.

This paper is based upon a study of the records of 182 cases of subphrenic abscess and on a perusal of the literature. The subject is so extensive that it would be impossible to discuss all aspects in the space allotted, and no attempt will be made to do so. I propose to confine my remarks to certain features of interest or importance.

ANATOMICAL CONSIDERATIONS

For the purposes of this discussion the subphrenic region will be taken to include the area from the diaphragm above to the transverse colon and mesocolon below. This region is divided into suprahepatic and infrahepatic compartments by the liver. The suprahepatic compartment is divided into right and left portions by the falciform ligament, and the infrahepatic compartment is similarly divided by the ligamentum teres and ligamentum venosum.

Our knowledge of the arrangement of the ligaments attached to the liver requires clarifying. It is a common misconception that the liver is suspended from the dome of the diaphragm by the coronary, triangular, and falciform ligaments, but in fact the coronary and triangular ligaments are fixed to the posterior aspect of the liver, which, like most other abdominal viscera, is attached by its ligaments to the posterior abdominal wall, formed at this level by the diaphragm.

The liver is normally kept in contact with the dome of the diaphragm not by ligamentous attach-

ments but by the mutual attraction of two closely applied serous surfaces separated only by a capillary layer of fluid. If these surfaces are separated by air, the liver falls away from the diaphragm as though it were hinged posteriorly about its bare area, the hinge being formed by the coronary and triangular ligaments. These points are illustrated in Plates I to III.

The confusion in the topography and classification of the subphrenic spaces is largely due to these misconceptions in respect of the attachments of the ligaments of the liver. In my opinion there are five intraperitoneal and two extraperitoneal spaces. The latter will not be considered. Of the five intraperitoneal spaces, two are situated on the right side and three on the left.

The two spaces on the right side are, I think, best called suprahepatic and infrahepatic, and they correspond with those described by Barnard (1908) as the right anterior and right posterior intraperitoneal spaces. Many surgeons, however, believe that there are two spaces above the right lobe of the liver, anterior and posterior, separated by the right triangular ligament. This view is held by Ochsner and Graves (1933), Ochsner and De Bakey (1938), Delario (1934), Faxon (1940), Clagett and Tinney (1944), Thorek (1947), and many other authorities. Lehman and Archer (1937) and Overholt and Donchess (1935) recognize two spaces above the liver on the right side, but believe that their separation is not of much practical importance. On the other hand Fifield and Love (1926) dispute, and Mitchell (1940) denies, the existence of the right posterior superior space.

A study of the anatomy of the parts clearly shows that there is only one suprahepatic space on the right side. This space is bounded posteriorly by the superior layers of the coronary and right triangular ligaments, and these layers separate the superior from the posterior surface of the liver. The inferior layers of the coronary and right triangular ligaments skirt the renal and suprarenal impressions on the right lobe of the liver, and so separate the posterior from the inferior surface of

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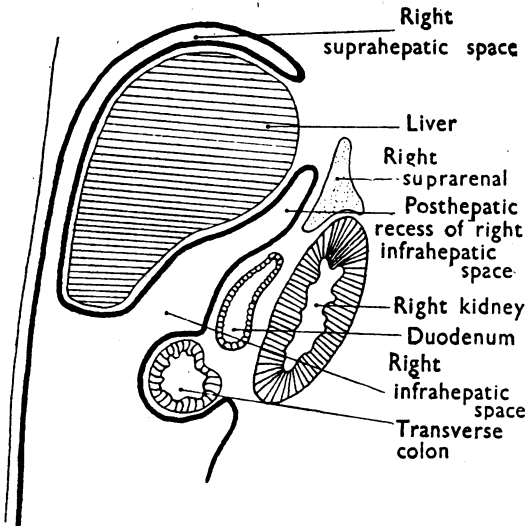


FIG. 1.—Sagittal section through abdomen to right of median plane (diagrammatic) showing right suprahepatic and right infrahepatic subphrenic spaces (called right subphrenic and right subhepatic spaces respectively by Mitchell). This, and Figs. 2 and 3, are redrawn from Mitchell's article in the *British Journal of Surgery* (1940) by kind permission of author and publisher.

the viscus and limit the postero-superior extremity of the right infrahepatic space. The right infrahepatic space is frequently called Morison's pouch. Its postero-superior extremity is situated between the right lobe of the liver and the upper pole of the right kidney, and is regarded by Mitchell as

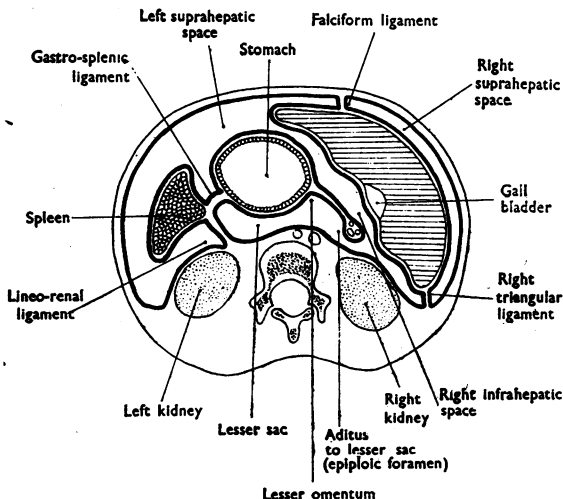


FIG. 2.—Horizontal section through upper part of abdomen (diagrammatic) showing the five subphrenic spaces. (After Mitchell, 1940.)

a retrohepatic recess of the right subhepatic space. This portion of the right infrahepatic space is that which, in my opinion, has been described incorrectly by many workers as the right posterior superior intraperitoneal space. These features are illustrated in Figs. 1 and 2.

On the left side a large potential space separates the diaphragm from the left lobe of the liver, the fundus of the stomach, and the spleen. Though

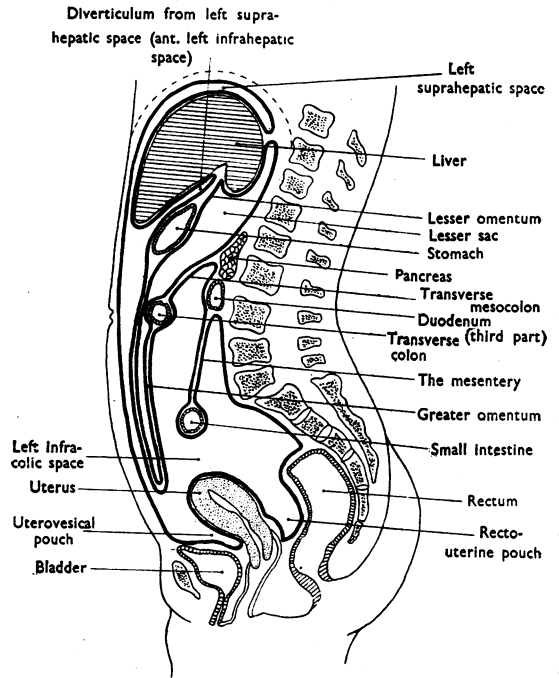


FIG. 3.—Sagittal section through abdomen to left of median plane (diagrammatic) showing left suprahepatic space (left subphrenic space of Mitchell), left anterior infrahepatic space (subhepatic recess of left subphrenic space of Mitchell), and left posterior infrahepatic space. (After Mitchell, 1940.)

the left lobe of the liver is related only to a half or less of the left dome of the diaphragm, this space is best called the left suprahepatic space in order that the terminology may conform with that used on the right side. Mitchell calls this space the left subphrenic space, a name which, though more accurate anatomically, is confusing in view of the wider significance of the term subphrenic as applied to infection in the region below the diaphragm.

There are two infrahepatic spaces on the left side, anterior and posterior. The left anterior infrahepatic space lies between the left lobe of the liver above and in front, and the stomach and lesser

TABLE I

FREQUENCY OF INVOLVEMENT OF THE VARIOUS INTRAPERITONEAL SUBPHRENIC SPACES IN 123 CASES OF SINGLE-SPACE INFECTION AND 21 CASES OF MULTIPLE-SPACE INFECTION

Space involved	Single-space infection	Multiple-space infection	Total
Right suprahepatic ..	71	14	85
Right infrahepatic ..	17	10	27
Left suprahepatic ..	23	10	33
Left infrahepatic, anterior	9	7	16
Left infrahepatic, posterior	3	4	7

omentum below and behind. It is in wide communication anteriorly with the left suprahepatic space, of which it is regarded by Mitchell merely as a subhepatic extension. The left posterior infrahepatic space comprises the lesser sac of the peritoneum. It extends much higher up between the liver and diaphragm than does the right infrahepatic space. These spaces are illustrated in Figs. 2 and 3. The frequency of abscess formation in the various intraperitoneal spaces in 144 cases in this series in which the spaces involved were identified is illustrated in Table I.

THE DIFFERENCE BETWEEN INFECTION SITUATED ABOVE AND BELOW THE LIVER

In respect of their clinical course, their signs, symptoms, and complications, suprahepatic and

infrahepatic infections are very different, especially on the right side. A study of Table II shows that, in the present series, symptoms and signs were most commonly present both in the chest and in the abdomen, wherever the infection was located, but that when they were confined to the chest the infection was always suprahepatic. On the right side symptoms and signs were more common in the chest than in the abdomen when infection was suprahepatic, but when it was situated below the liver they were more common in the abdomen. The figures on the left side are too small to be significant.

The lower part of Table II indicates that on each side suprahepatic infection often gives rise to thoracic complications, but that infrahepatic infection much less commonly does so, especially on the right side. The left anterior infrahepatic space was the site of abscess formation in all cases in which serous effusion or empyema complicated infection below the diaphragm on the left side. Infection occurring in this space has easy access to the diaphragm by passing forwards between the liver and stomach, so that thoracic complications might be expected to occur in a proportion of cases when this space is involved.

Perforation of the diaphragm occurred in 15 of the 123 cases shown in Table II. In all these the abscess was suprahepatic, in 10 it was on the right side, and in 5 it was on the left. Four other abscesses in this series also perforated the dia-

TABLE II

CLINICAL FEATURES OF INFECTION IN 123 CASES OF INTRAPERITONEAL SUBPHRENIC ABSCESS INVOLVING ONE SPACE ONLY

Clinical features	Right suprahepatic infection	Right infrahepatic infection	Left suprahepatic infection	Left anterior infrahepatic infection	Left posterior infrahepatic infection	Total
Thoracic symptoms and signs only	16	—	3	—	—	19 (16%)
Abdominal symptoms and signs only	6	6	2	2	1	17 (14%)
Both thoracic and abdominal symptoms and signs	44	6	16	5	2	73 (59%)
Symptoms and signs not recorded	5	5	2	2	—	14 (11%)
Total	71 (58%)	17 (14%)	23 (19%)	9 (7%)	3 (2%)	123
Serous effusion	17	1	12	2	—	32 (26%)
Empyema	20	—	5	1	—	26 (21%)
Suppurative pneumonitis	8	—	—	—	—	8 (7%)
Died	19	7	9	8	2	45 (37%)

phragm, and in all these infection was present above the liver.

The high mortality of infrahepatic infection is worthy of note. This is due to its association with serious abdominal disease or complications.

SUBPHRENIC ABSCESS ON THE LEFT SIDE

In the present series of 182 cases of subphrenic abscess 119 (65.4 per cent) occurred on the right side and 50 (27.5 per cent) on the left. In 10 cases (5.5 per cent) the abscess was bilateral, and in three the site of the abscess was not stated. These figures are shown in Table III.

TABLE III
SIDE INCIDENCE OF SUBPHRENIC INFECTION

Side	Number	%
Right	119	65.4
Left	50	27.5
Bilateral .. .	10	5.5
Not recorded ..	3	1.6
Total	182	—

Subphrenic abscess occurs more commonly on the left side than is generally realized, and when it does so its clinical features are of considerable interest. Carter (1939) states, "When the subject of subphrenic abscess is being considered, as a rule only those abscesses that occur beneath the right half of the diaphragm are thought of and rarely, if ever, is the possibility of there being an abscess beneath the left half of the diaphragm given any consideration. In other words, to the majority of physicians subphrenic abscess really means right subphrenic abscess." Carter (1939) maintains that the ratio of subphrenic abscess on the right and left sides is one to three or four. The ratio in the present series is even higher than this. Of 1,531 cases of subphrenic abscess collected by Ochsner and De Bakey (1938) the left side was involved in 27.6 per cent. Neuhof and Schlossmann (1942) reported 33 cases of abscess on the left side compared with 51 cases on the right side, and stress the slight attention paid in the literature to left-sided infection.

Both Carter (1939) and Neuhof and Schlossmann (1942) point out certain differences between abscesses occurring on the right and left sides, and the present study supports most of their contentions. Suprahepatic abscesses on the right side

are confined inferiorly by the massive right lobe of the liver, while those on the left side are bounded by the much more mobile spleen, stomach, and left lobe of the liver. For these anatomical reasons elevation of the diaphragm occurs less commonly on the left side, and other thoracic changes are also less common. During a radiological examination a slight elevation of the diaphragm is more likely to be overlooked on the left side than on the right. A slight rise on the right side accentuates the normal difference in height between the two domes of the diaphragm, whereas a similar rise on the left side diminishes the difference and makes it less obvious. Furthermore, gas in a left-sided subphrenic abscess is liable to be mistaken for gas in the stomach or splenic flexure of the colon. For these reasons radiological diagnosis is more difficult on the left side than on the right.

Left-sided abscesses are more likely to be multilocular and so are more difficult to drain than those on the right side. This is because several viscera lie beneath the left dome of the diaphragm, whereas only one lies below that on the right side. Another difference of some importance is that subphrenic abscess on the left side occurs more commonly in consequence of malignant disease than is the case on the right side. In the present series carcinoma of the stomach, colon, or pancreas was the cause of 12 per cent of abscesses on the left side but of only 4.2 per cent of those on the right. Neuhof and Schlossmann (1942) found breaking down retroperitoneal sarcomas in 4 of their 33 cases of left-sided subphrenic abscesses. All these matters are important because they contribute to the high mortality of left-sided abscesses, 50 per cent in this series as against 31 per cent for abscesses on the right side. Neuhof and Schlossmann give the corresponding mortality figures as 75 per cent and 35 per cent.

MULTIPLE-SPACE ABSCESSSES

By multiple-space abscess is meant one affecting two or more of the anatomical subphrenic spaces. There were twenty-three of these (12.1 per cent) in the present series, all except one of which involved two spaces. In one three spaces were infected. An abscess may be multilocular and yet be confined to a single space. Such an abscess is shown in Plate Xa and b.

In 10 cases (5.4 per cent) the abscess was bilateral and in 13 it was unilateral. Two abscesses affected an intraperitoneal and an extraperitoneal space (both unilateral) and 21 affected more than one intraperitoneal space.

The multiple-space abscesses were much more fatal than the single-space ones. This is shown in Table IV, which indicates the mortality of intraperitoneal infections.

TABLE IV
MORTALITY IN SINGLE-SPACE AND MULTIPLE-SPACE
INFECTION (INTRAPERITONEAL)

	Number	Died
Single-space infection ..	143	45 (32%)
Unilateral multiple-space infection ..	11	6 (55%)
Bilateral multiple-space infection ..	10	9 (90%)

Seven of the 10 bilateral cases were due to perforations of peptic ulcers, and 9 of the patients died. So high a mortality is worthy of note. An important cause for this was failure to diagnose the bilateral nature of the infection and therefore to establish adequate drainage. Only 6 of the abscesses were drained on both sides. Two were drained on the right side only, and in 2 no drainage was performed at all.

If drainage of a subphrenic abscess is not followed by the patient's recovery the presence of infection on the opposite side must be considered.

"PRIMARY" SUBPHRENIC ABSCESS

By "primary" subphrenic abscess is meant one in which no causal lesion is found. There were 17 of these (9.3 per cent) in the present series; 13 on the right side and 4 on the left. In Faxon's 111 cases (1940) the incidence of primary abscess was 26 per cent. All the abscesses in the present series were intraperitoneal, and one of them on the left side affected two spaces.

The bacteriology, clinical features, and complications were much the same as those of other types of abscess. In 13 the initial symptom was acute abdominal pain suggestive in character of acute appendicitis, perforated peptic ulcer, acute cholecystitis, or infarction of the spleen, and in several of these a swelling developed in the hypochondrium. One developed pain in the left lower chest. Two presented with loin pain, simulating a perinephric abscess, and 2 with pleural effusion on the left side. Four abscesses contained gas, and in 3 of these a bronchial fistula was present. Six of the 17 developed suppurative thoracic complications.

Four patients died, making a mortality of 23.5 per cent. This is much lower than that for the whole series, as might be expected, because in

these primary abscesses the subphrenic infection is the only disease present. Of the 4 cases that died 2 were neither diagnosed nor drained, 1 developed an empyema in consequence of perforation of the diaphragm, and 1 developed meningitis.

CHRONIC SUBPHRENIC ABSCESS

Barnard (1908) and Whipple (1926) have both described a variety of subphrenic abscess characterized by a "slow, insidious" onset. Beye (1932) writes that subphrenic abscess may be "almost unbelievably chronic," and that thoracic complications may develop suddenly or insidiously at any time during the course of the disease and produce a confused picture resembling that of tuberculosis, bronchiectasis, or empyema. Indeed, the diagnosis may be established only when an empyema is drained and a communication through the diaphragm is found at operation.

Lewenstein (1946) states that symptoms may be delayed for weeks or months after the onset of the condition and that, though apparent recovery may ensue, there is never complete restoration of health. Weakness, lassitude, and pain of vague and varying character persist, but localizing symptoms are minimal or absent. He notes that the diagnosis may suddenly become evident, as complications develop, or alternatively in their absence that the abscess may long remain unrecognized and occasion only the general picture of prolonged sepsis. Lehman and Archer (1937) also discuss the possibility of a prolonged latent period.

A chronic abscess is defined as one that has been present for six months or more prior to drainage, or to death of the patient in the absence of drainage. A primary abscess is considered to begin at the time of onset of symptoms, and a secondary one at the time of the causal condition. There were 16 chronic cases in this series, and others have been reported by a number of observers. Nine of the abscesses were present for a year or more before drainage, and 2 were present for ten years.

One abscess was "primary," 1 was secondary to actinomycosis of the lung, and 14 followed abdominal operation or suppuration. The aetiology and location of the abscess were similar in the acute and chronic varieties. Two of the chronic abscesses were bilateral.

The "primary" abscess presented with a pleural effusion, and the actinomycotic one with an empyema. The abscesses which followed an abdominal operation or infection manifested themselves clinically in one of two ways. In 5 of them

symptoms were present from the outset, but their significance was not appreciated for months or years. In 9 others no symptoms were present at the outset, and a latent period ensued varying from fourteen days to nine years, during which the patient was apparently well. Thereafter the onset of symptoms when it occurred was abrupt in 5 cases; in 4 it was ill-defined and vague.

TABLE V

THORACIC COMPLICATIONS FOLLOWING ACUTE AND CHRONIC SUBPHRENIC INFECTION

	Acute abscess	Chronic abscess
Serous effusion	42 (26%)	5 (31%)
Empyema or pyopneumothorax	30 (18%)	4 (25%)
Suppurative pneumonitis ..	11 (7%)	3 (19%)
Bronchial fistula	16 (10%)	4 (25%)

Pain, either alone or in combination with fever, malaise, or productive cough, was the initial feature ten times, and pleural effusion twice. Empyema, productive cough, external rupture of the abscess, and obstructive jaundice were occasionally primary manifestations. Where productive cough is a feature of the onset of symptoms the picture may closely resemble that of an acute lung abscess, being similar in respect of the large quantity of foul, purulent sputum which may suddenly be expectorated.

It is noteworthy that 14 of the 16 patients showed localizing symptoms or signs, and that elevation of the diaphragm, often accompanied by other changes, was demonstrated in all twelve patients on whom radiological examination was performed.

By comparison with acute abscesses the incidence of pleural effusion and empyema was slightly higher and of suppurative pneumonitis and bronchial fistula markedly higher in the chronic cases, as shown in Table V.

Furthermore, rupture into a bronchus or the stomach, or on to the surface of the body, occurred in 37.5 per cent of chronic abscesses as compared with only 9.9 per cent of acute ones. It is evident that undrained abscesses point to a surface and tend to evacuate themselves there.

All the chronic abscesses were drained, and 37.5 per cent of the patients died. The mortality of acute cases which were drained in this series was 25.2 per cent. The chronic abscess appears, therefore, to be a somewhat more lethal condition than the acute variety, though the figures are too small to be conclusive.

SPREAD OF INFECTIVE PROCESSES WITHIN THE ABDOMEN

In 152 cases of the present series the abscess followed abdominal inflammation. In 98 of these (64.5 per cent) the infection spread from an adjacent focus, in 15 (9.9 per cent) the abscess was a residual one following general peritonitis, and in 4 (2.6 per cent) there was pathological evidence that infection had spread from the appendix via the paracolic gutter.

In these three groups, comprising 117 of the 152 cases (76.9 per cent), the method of spread of infection was an intraperitoneal one. In 4 patients spread of infection from a distant focus took place via the portal vein and resulted in suppurative pylephlebitis with multiple liver abscesses.

In the remaining 31 patients the infection was remote in the abdomen and there was no evidence as to how it reached the subphrenic space. In no case was there evidence of lymphatic spread, which might be expected to occasion a regional adenitis but not an abscess within the peritoneal cavity unrelated to the glands.

The great majority of abscesses are due to abdominal infections arising for the most part in the vicinity of the subphrenic region. Some are residual in character and follow general peritonitis, and a few are due to suppurative pylephlebitis. These findings are summarized in Table VI.

TABLE VI

INTRA-ABDOMINAL SPREAD OF INFECTION TO SUBPHRENIC REGION IN 152 CASES OF SUBPHRENIC ABSCESS SECONDARY TO INTRA-ABDOMINAL INFLAMMATION

Method of spread of infection	Number	%
Neighbouring infection ..	98	64
Residual abscess following general peritonitis	15	10
Spread via paracolic gutter ..	4	3
Spread via portal vein	4	3
Method of spread not determined	31	20

The mechanism of spread of infection to the suprahepatic region from distant parts of the peritoneal cavity has been variously explained. In my opinion infection is usually carried to this region by the movement of infected peritoneal fluid influenced by the force of gravity or by the differing hydrostatic pressures within the various parts of the peritoneal cavity. In the recumbent position, which is usually assumed after an abdominal catastrophe or during sleep, the region where the diaphragm joins the posterior abdomi-

nal wall is a low point to which fluid will naturally gravitate and from which it is soon spread by the constant movement occurring between the diaphragm and its related viscera. The effect of this movement can readily be observed by placing a moistened cover-slip on a microscope slide. If a drop of dye is placed nearby and the cover slip moved to and fro on the slide, the dye will rapidly spread between the two surfaces. Mass movement of fluid occurs in serous cavities in accordance with the laws of gravity. This has been proved in the case of pleural effusions by Blair (personal communication), who has shown by *x*-ray studies that the pleural fluid moves quickly to new positions with changes of posture. It is legitimate to assume that peritoneal fluid behaves similarly before adhesions have formed.

Overholt (1931) has shown that in the dog the pressure in the suprahepatic region is lower than in the general peritoneal cavity and varies with the phase of respiration. When respiration is quiet the pressure falls during inspiration and rises during expiration. These findings have been confirmed by Banyai (1946), though they are in conflict with those of others. Overholt (1931) has also shown that the pressure in any part of the peritoneal cavity varies with posture, being greater in the dependent areas and less elsewhere. This has been confirmed for the experimental animal by Lam (1939) and Rushmer (1946) and for man, after abdominal operations have been performed, by Drye (1948). The last worker found that the pressure was the same in all parts of the peritoneal cavity in the supine position, but that in the erect position the pressure in the lower abdomen increased to nearly three times that in the upper abdomen. In this respect the abdomen follows the laws obeyed by a closed box with partly rigid and partly flexible walls, to which it has been likened by Wildesgans (1923), Krause (1927), and Hitzenberger (1929). The magnitude of these pressure changes in the dog is illustrated in the following experiment performed by Overholt (1931):

Position of animal	Cannula in epigastrium	Cannula in lower abdomen
Horizontal	Minus 47 mm. of water	Minus 25 mm. of water
Head up	Minus 58 mm. of water	Plus 103 mm. of water
Head down	Plus 49 mm. of water	Minus 127 mm. of water

It may be, as suggested by Overholt and Donchess (1935), that these pressure differences are sufficient

to produce in the sitting position an upward flow of peritoneal fluid to the subphrenic space despite the force of gravity. It would thus appear that whatever may be the position, whether horizontal or upright, the forces suffice to move peritoneal fluid to the subphrenic region, and it matters little in what position the patient is nursed.

Spalding (1943) postulates that a suprahepatic pneumoperitoneum of low relative pressure is of importance in aspirating infected fluid from the infrahepatic to the suprahepatic region. He points out the frequent association of subphrenic abscess with conditions causing pneumoperitoneum. My own view is that pneumoperitoneum has not the significance assigned to it by Spalding, and that if it plays a part in the production of subphrenic infection it does so by opening up the subphrenic spaces. Perforated peptic ulcer, though it gives rise to pneumoperitoneum, also occasions gross contamination of the peritoneal cavity; furthermore subphrenic abscess occurs in the absence of perforation, or laparotomy, or pneumoperitoneum.

The force of capillarity is probably insufficient to cause any significant movement of infected fluid to the subphrenic region, and moreover it cannot act unless there is an intersurface between fluid and gas or between two fluids of differing constitution.

SUBPHRENIC ABSCESS ASSOCIATED WITH LIVER ABSCESS

Liver abscess was present in 19 of the 182 cases (10.4 per cent), and was frequently multiple. In 2 cases it appeared to be a primary disease, in 9 it was concomitant with the subphrenic abscess, and in 8 it appeared to be secondary to the subphrenic abscess. Suppurative pylophlebitis was present in 16 of the 17 cases in the second and third categories, and suppurative cholangitis in 1.

The clinical features which suggest liver abscess are rigors (5), jaundice (5), and enlargement (6) or tenderness (4) of the liver, but all these are inconstant. Fifteen of the 19 cases with liver abscess died, making a mortality of 79 per cent.

About 1 case in every 10 of subphrenic abscess is associated with liver abscess, and 4 out of 5 of such cases die. The presence of a liver abscess should always be considered when drainage of a subphrenic abscess is not attended by recovery of the patient.

THORACIC COMPLICATIONS AND THE SPREAD OF INFECTION BETWEEN ABDOMEN AND CHEST

From a study of the thoracic complications of subphrenic abscess it is evident that serous pleural

effusion should be considered separately from intrathoracic suppurations.

A serous effusion, either clear or cloudy in appearance, was proved to be present by aspiration, operation, or necropsy in 47 cases (25.8 per cent). In 12 other cases the presence of an effusion was suggested by x-ray examination, but was not otherwise confirmed. Serous effusion is thus a common sequela of subphrenic infection.

It is noteworthy that in 6 cases the effusion was contralateral and in 2 it was bilateral. It is important to know that contralateral effusions may occur, because of the difficulty they may occasion in diagnosis.

In both bilateral cases malignant disease was present, in the stomach or the caecum, and perforation of the bowel occurred, with fatal peritonitis. In neither case was the subphrenic abscess recognized or drained.

In all the cases with contralateral or bilateral effusion the subphrenic abscess was unilateral, and in 7 of the 8 cases it was situated on the right side. In 6 cases the abscess was intraperitoneal, in one extraperitoneal, and in one both intra- and extraperitoneal.

In 20 of the 47 cases the pleural fluid was examined bacteriologically. In 17 of these no organisms were demonstrated either in films or in cultures. In 1 case organisms were seen in films, but cultures were sterile. In only 2 cases did organisms grow in culture.

Examination of the protein content in 5 cases, and of the cellular content in 15, suggested that the nature of the pleural fluid was inflammatory. The protein content varied between 1.2 per cent and 3.3 per cent. The chief cellular constituents were red cells, neutrophils, and lymphocytes. A differential cell count was made in 12 cases. In 8 of these, neutrophils predominated over lymphocytes, and in 3 lymphocytes predominated over neutrophils. In 1 case the two types of cell were present in about equal proportions.

No cause within the chest was found to account for the serous effusion in 39 of the 47 cases. In 8 cases a possible cause was present in the lung or pleura, such as fibrino-purulent pleurisy or suppurative pneumonitis. It is my belief that where no obvious cause can be found in the chest the serous effusion is an expression of the reaction of the subpleural vessels to a neighbouring focus of infection. These vessels are subject to inflammatory vasodilatation, and the outpouring of serous fluid into the pleura is analogous to the oedema which occurs around an acute inflammatory focus elsewhere. An effusion of similar

character occurs in a joint when acute inflammation develops in its neighbourhood.

When suppuration in the chest follows in consequence of a subphrenic abscess the cause is usually obvious. This is illustrated in Table VII, which analyses the cases of empyema and suppurative pneumonitis, and is in marked contrast with the findings in the case of serous pleural effusion.

TABLE VII

CAUSES OF EMPYEMA, PYOPNEUMOTHORAX, AND SUPPURATIVE PNEUMONITIS IN 182 CASES OF SUBPHRENIC ABSCESS

<i>Empyema and pyopneumothorax—38 cases (29%):</i>	
Transpleural drainage	12
Perforation of diaphragm	9
Penetrating wounds	6
Lung suppuration	2
Diagnostic aspiration	1
Open drainage of serous effusion	1
Following serous effusion	1
No cause found	6
<i>Suppurative pneumonitis—14 cases (8%):</i>	
Perforation of diaphragm	6
Empyema	3
Inhalation	3
No cause found	2

It is evident from Table VII that the three most important causes of suppuration within the chest were rupture of the abscess through the diaphragm, transpleural drainage of the abscess, and penetrating thoraco-abdominal wounds. The ill effects of transpleural drainage will be discussed later. It is noteworthy that in 19 cases a subphrenic abscess ruptured through the diaphragm. (Only 15 of these appear in Table VII.)

In only 1 case did a serous effusion become purulent. Empyema does not follow a serous effusion except as a rare event. There was no evidence to suggest that spread of infection through the lymphatic vessels of the diaphragm was an important or frequent cause of intrathoracic suppuration, though it may have determined its onset in some of the cases, relatively few in number, in which no apparent cause was found.

When subphrenic infection leads to suppuration within the chest the mortality is markedly influenced by the development of a bronchial fistula, consequent on the rupture into a bronchus of the subphrenic abscess or of a secondary empyema. The mortality in this series for all cases complicated by empyema, pyopneumothorax, and suppurative pneumonitis was 42 per cent. When a bronchial fistula was present the mortality was only

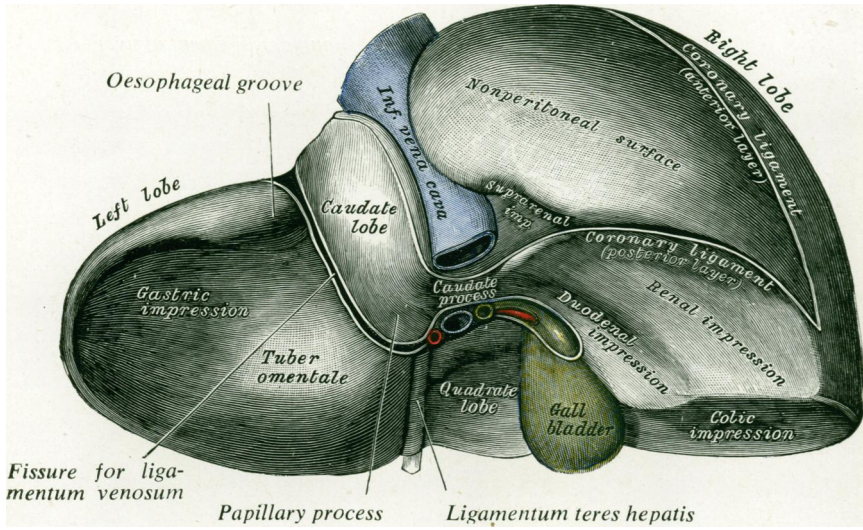


PLATE Ia.—Posterior and inferior surface of liver, showing attachments of coronary and right triangular ligaments. (From *Gray's Anatomy*, 28th edition, Fig. 1204.)

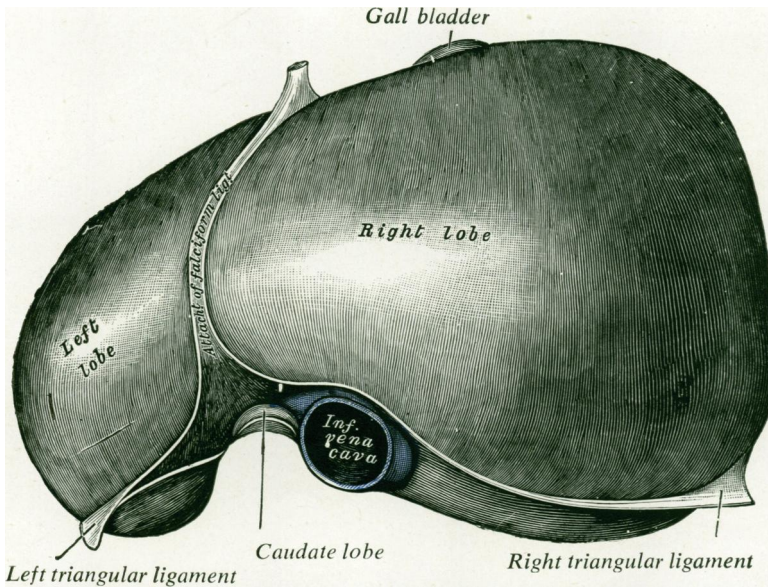
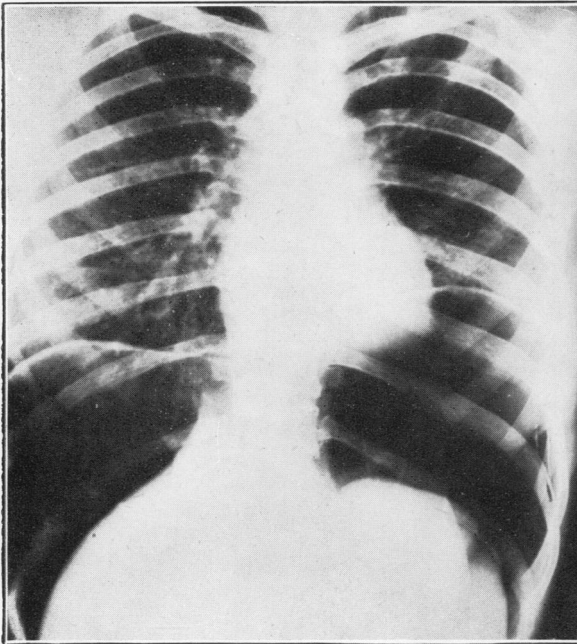
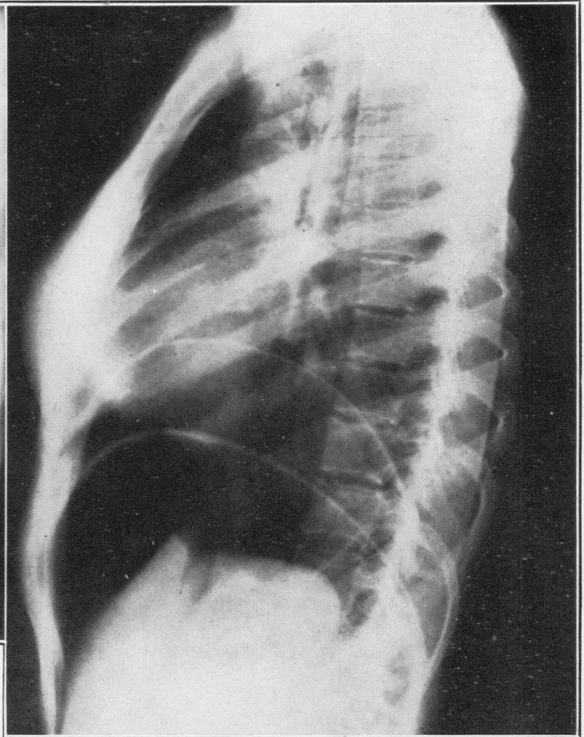


PLATE Ib.—Superior, anterior, and right lateral surfaces of liver, showing attachments of falciform ligament, superior layer of coronary ligament, and right and left triangular ligaments. (From *Gray's Anatomy*, 28th edition, Fig. 1203.) The two figures on this page are reproduced by kind permission of Messrs. Longmans, Green and Co.

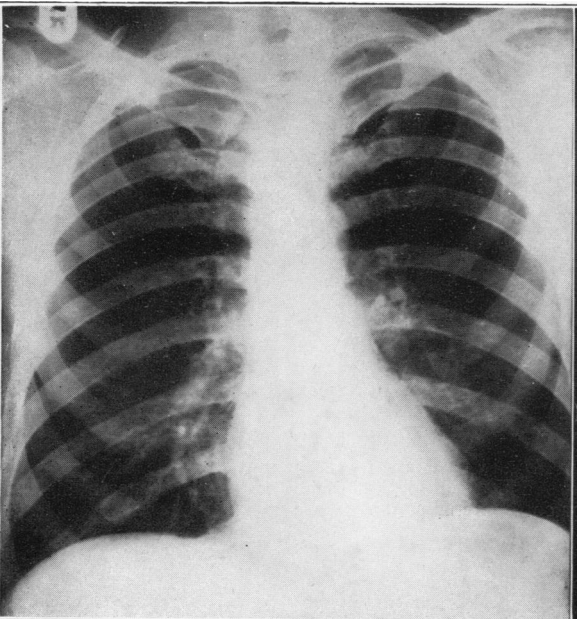


(a)

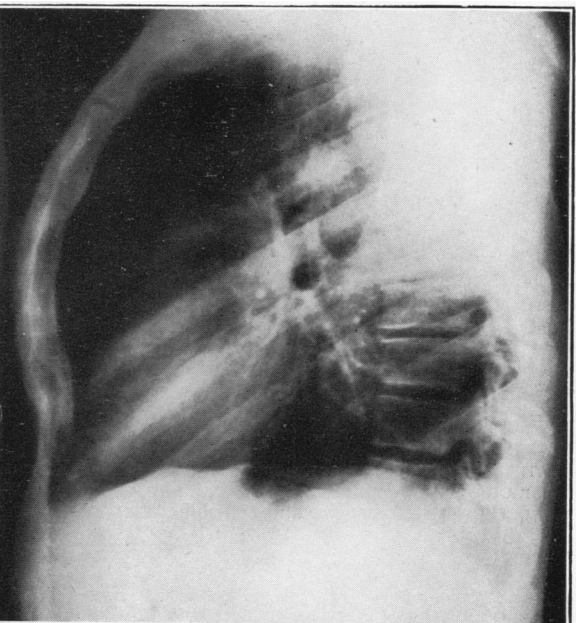


(b)

PLATE III.—(a) Postero-anterior film taken after induction of pneumoperitoneum to show how liver, stomach, and spleen fall away from the diaphragm. (b) Lateral film taken after induction of pneumoperitoneum to show how liver falls away from the diaphragm as though it were hinged posteriorly about its bare area.



(c)



(d)

PLATE III.—(c) Postero-anterior film of patient with left perinephric abscess, showing marked elevation of left diaphragm. (d) Lateral film of patient with left perinephric abscess.

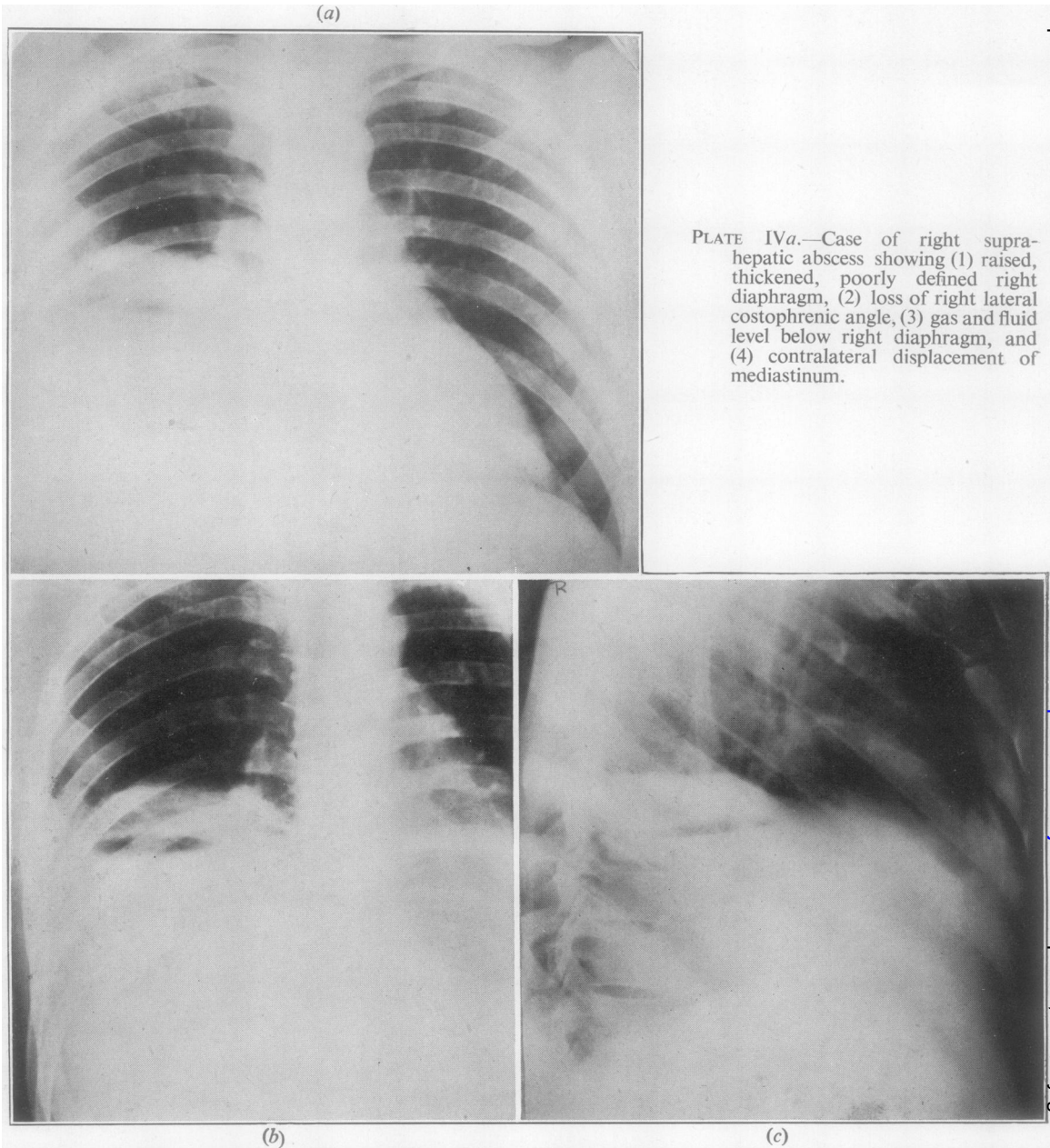
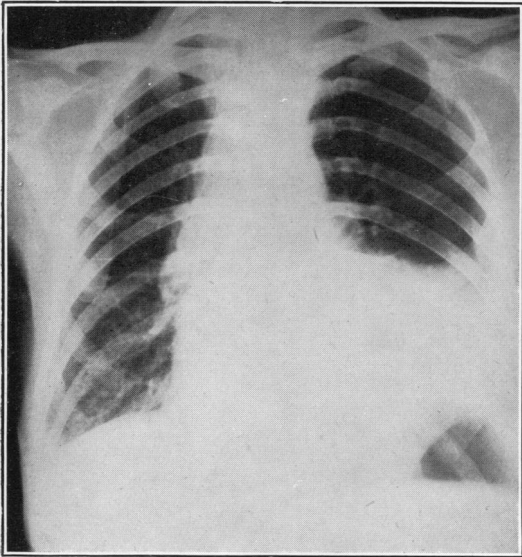
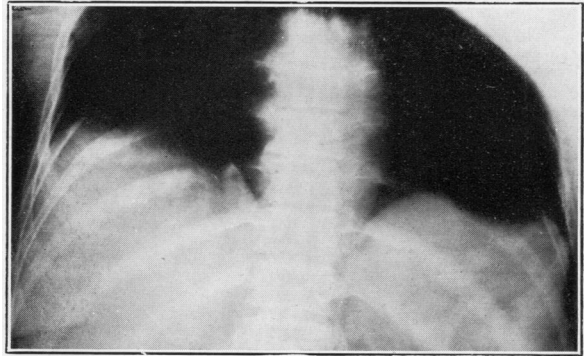


PLATE IV.—(b) Postero-anterior penetrating film. The gas and fluid level are more obvious, and the diaphragm is more clearly defined. (c) Lateral film showing (1) gas and fluid level situated posteriorly beneath highest portion of diaphragm, and (2) obliteration of posterior costophrenic angle.



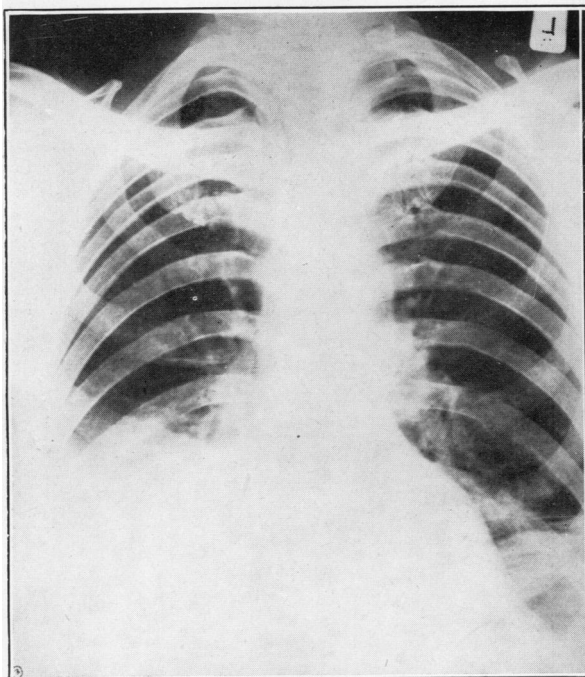
(a)



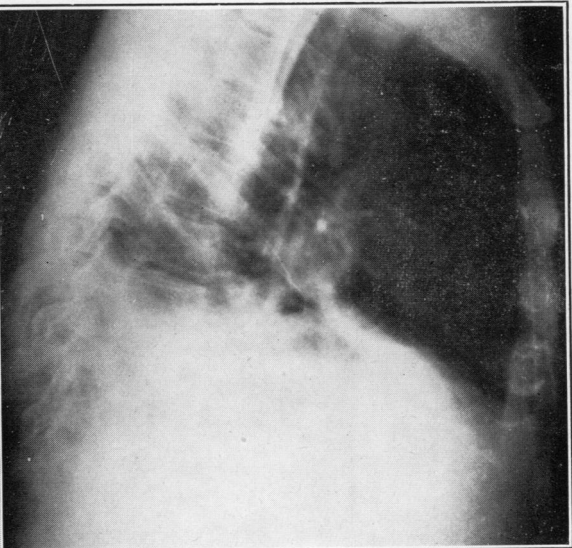
(b)

PLATE Va.—Case of left subphrenic abscess (space involved not known). Postero-anterior film showing (1) high left diaphragm, completely obscured by (2) uniform opacity at left base, probably due to pleural effusion (not proven), and (3) gas and fluid level below left diaphragm.

PLATE Vb.—Case of right infrahepatic abscess. Postero-anterior film showing (1) high, well-defined right diaphragm, and (2) normal right lung field.



(c)



(d)

PLATE V.—(c) Case of right suprahepatic abscess with pleural effusion. Postero-anterior film showing (1) raised, poorly defined right diaphragm, (2) loss of lateral costophrenic angle, due to pleural effusion, and (3) obscured cardiophrenic angle, probably due

to collapse of lower lobe. (d) Lateral film of same case showing (1) gas and fluid level below summit of right diaphragm. This is not visible in the postero-anterior film. (2) Loss of posterior costophrenic angle, due to pleural effusion. (3) Patchy opacities in lung field above posterior half of diaphragm, probably due to collapse of lower lobe and pneumonitis.

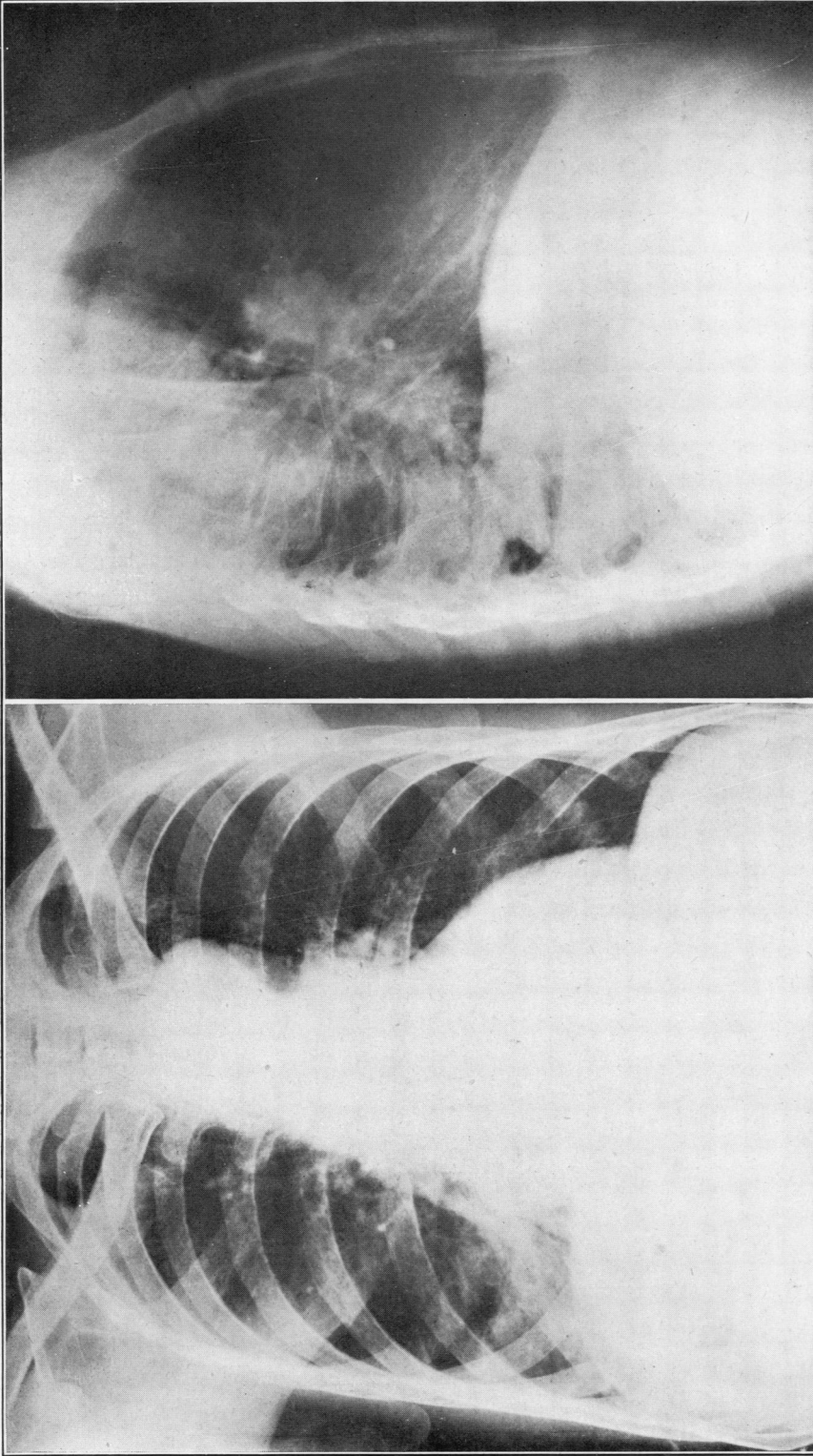


PLATE VI.—(a) Case of multiple space abscess involving the right suprahepatic and right infrahepatic spaces. Postero-anterior film showing (1) high, fairly well-defined right diaphragm, (2) clear lateral costophrenic angle, and (3) opacity at right base suggesting collapse of lower lobe. (b) Lateral film of same case showing (1) right diaphragm raised, especially posteriorly, (2) clear posterior costophrenic angle, (3) mottled opacities in lung field suggesting collapse of lower lobe, and (4) gas and fluid level beneath right diaphragm posteriorly (not visible in postero-anterior film).

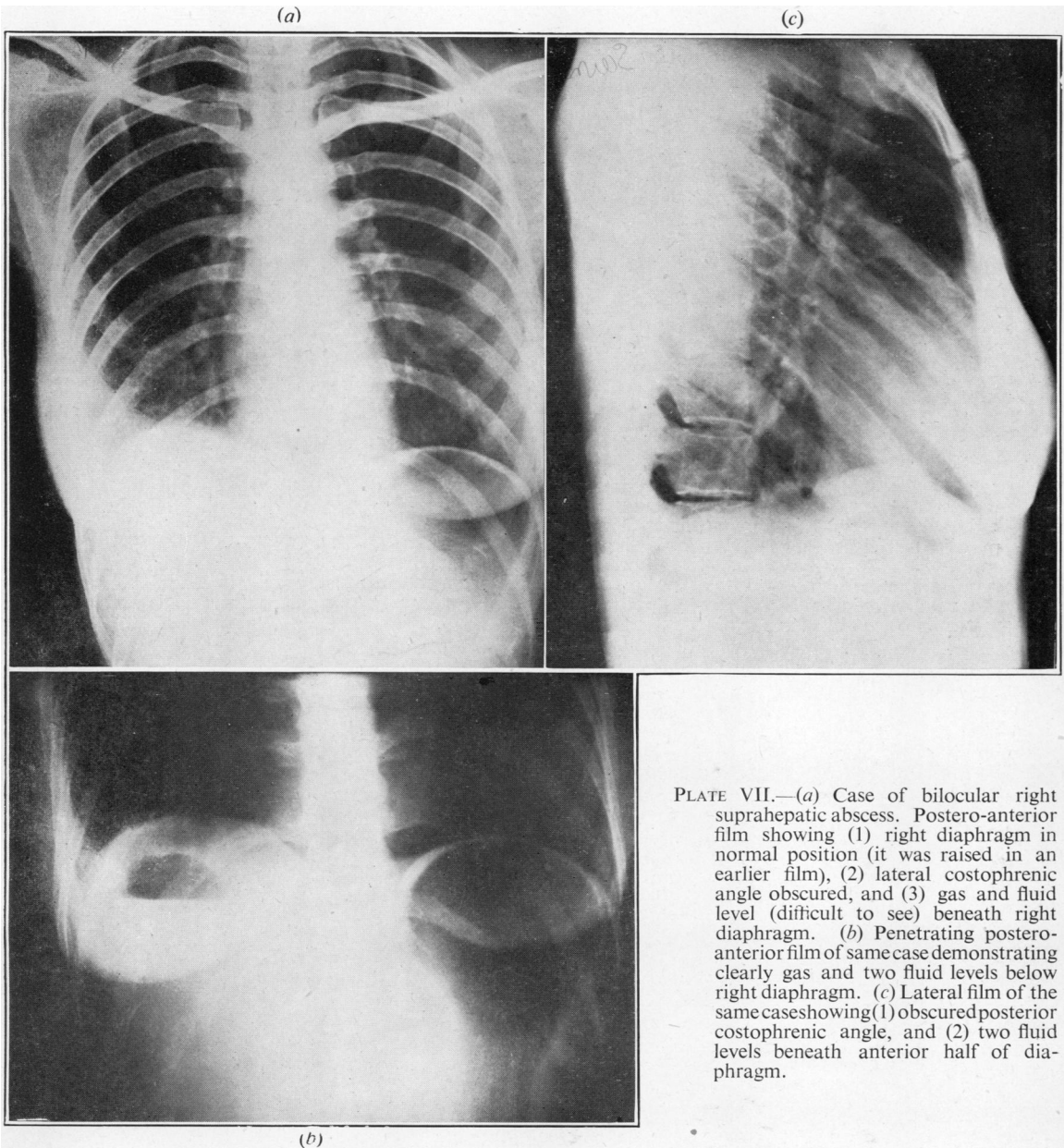


PLATE VII.—(a) Case of bilocular right suprahepatic abscess. Postero-anterior film showing (1) right diaphragm in normal position (it was raised in an earlier film), (2) lateral costophrenic angle obscured, and (3) gas and fluid level (difficult to see) beneath right diaphragm. (b) Penetrating postero-anterior film of same case demonstrating clearly gas and two fluid levels below right diaphragm. (c) Lateral film of the same case showing (1) obscured posterior costophrenic angle, and (2) two fluid levels beneath anterior half of diaphragm.

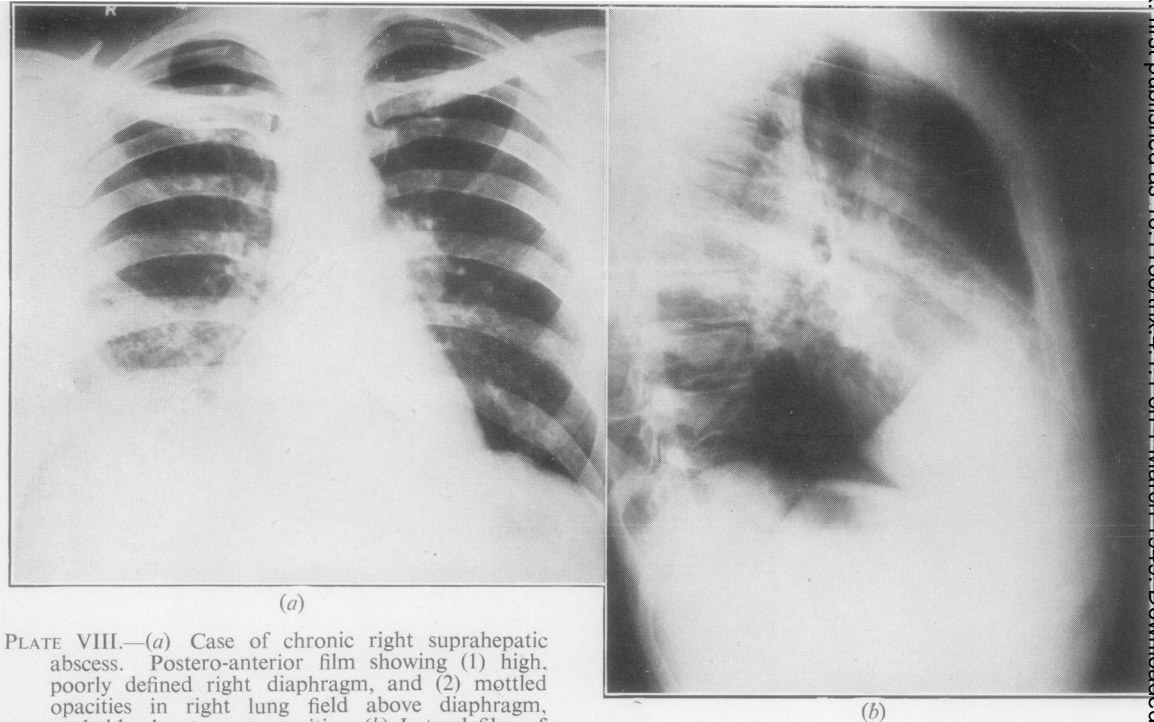


PLATE VIII.—(a) Case of chronic right suprahepatic abscess. Postero-anterior film showing (1) high, poorly defined right diaphragm, and (2) mottled opacities in right lung field above diaphragm, probably due to pneumonitis. (b) Lateral film of same patient showing (1) marked elevation of right diaphragm anteriorly, (2) opacity in lung field above highest part of diaphragm, probably due to pneumonitis, and (3) clear posterior costophrenic angle and well-defined posterior portion of diaphragm.

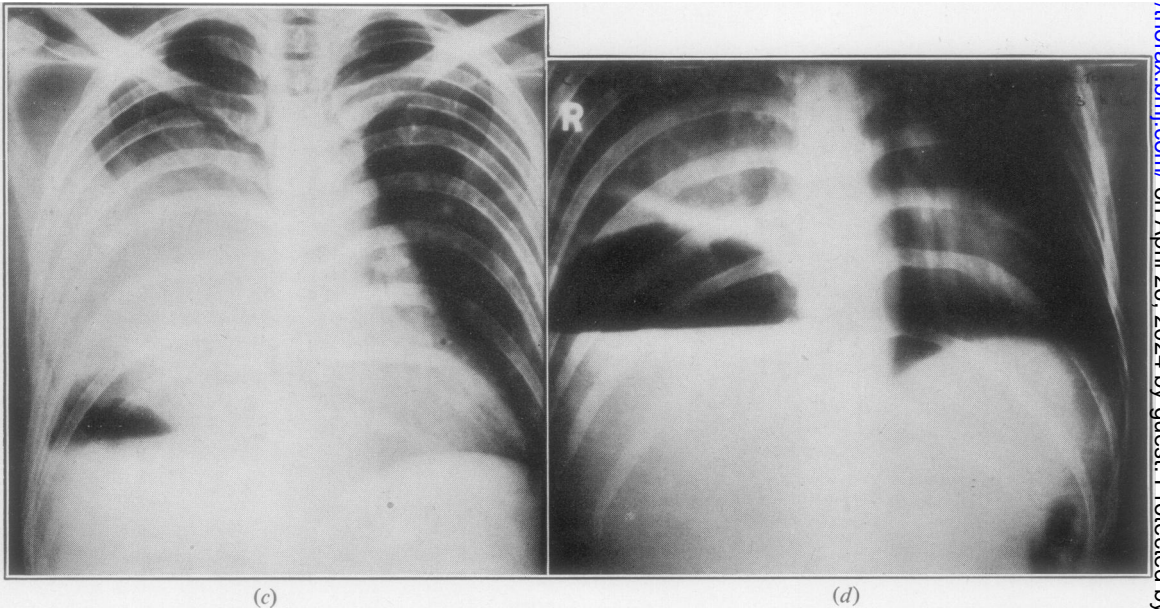
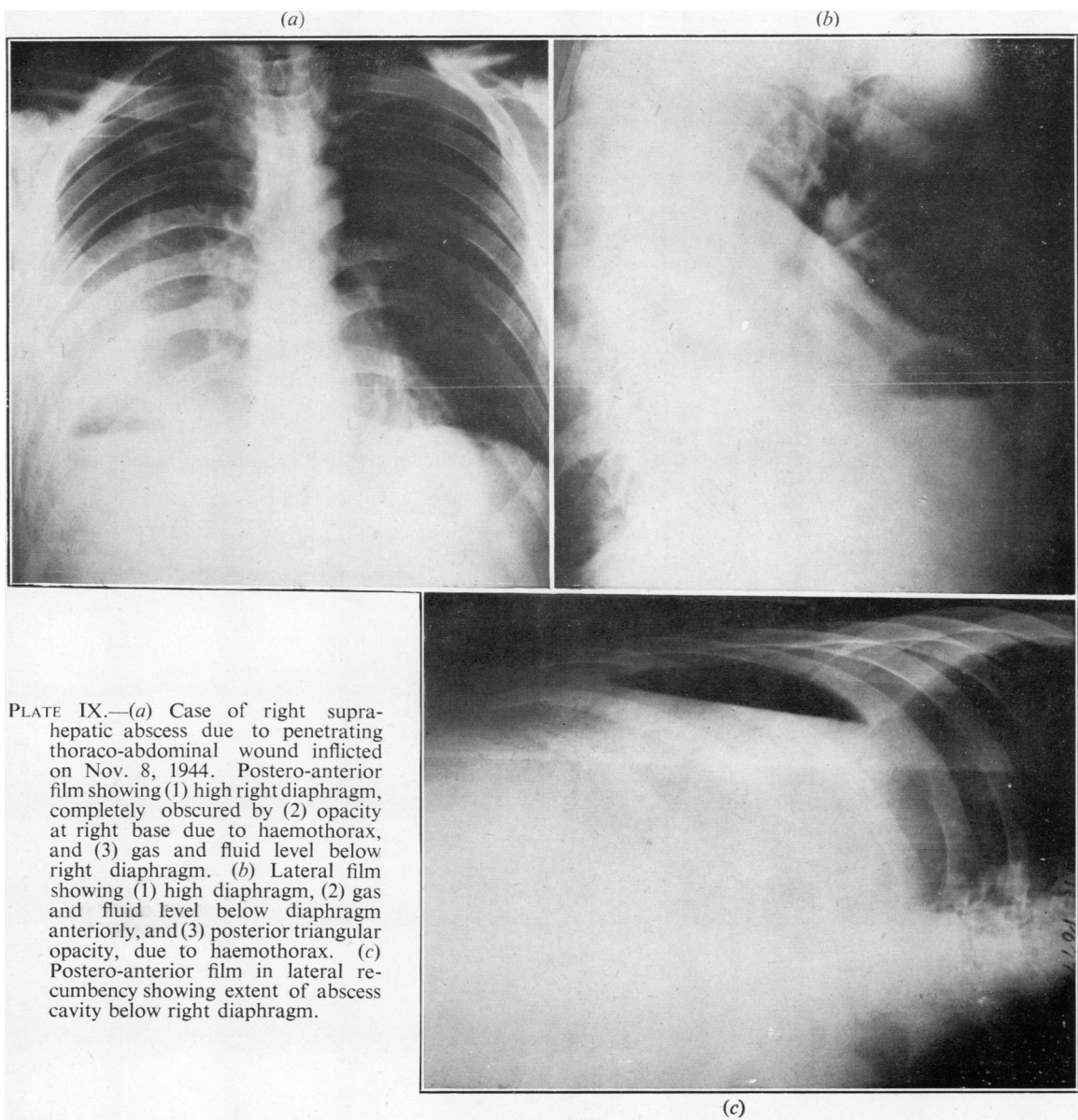
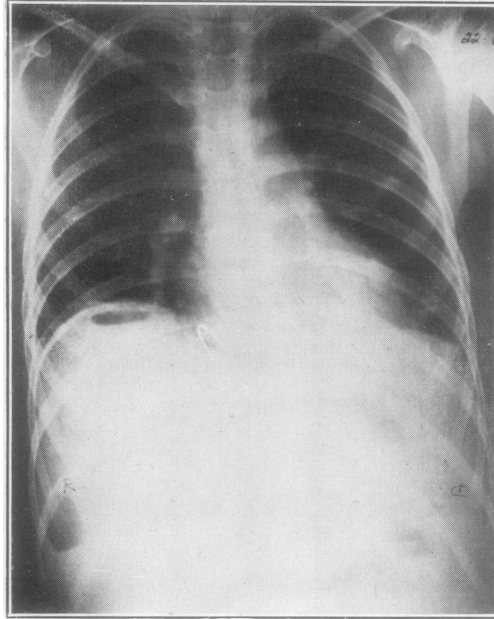
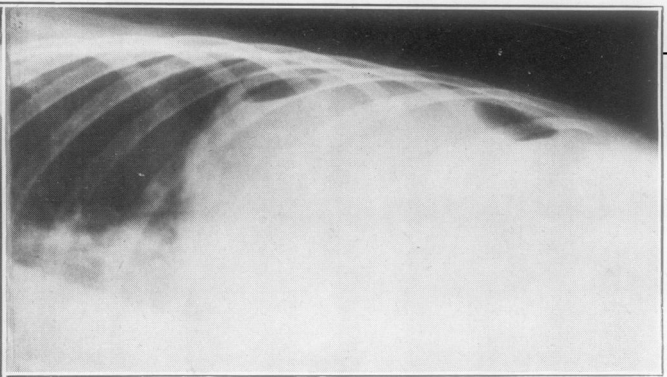


PLATE VIII.—(c) Case of right suprahepatic abscess due to penetrating thoraco-abdominal wound. Postero-anterior film showing (1) high right diaphragm, obscured by (2) opacity in right lung field due to pleural effusion, and (3) gas and fluid level beneath right diaphragm. (d) Penetrating basal film of same patient showing fluid level and diaphragm much more clearly defined than in (a).



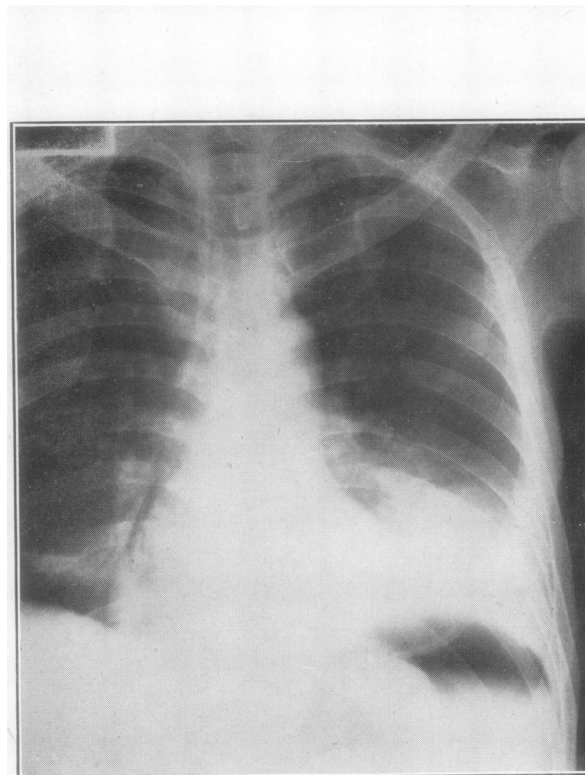


(a)



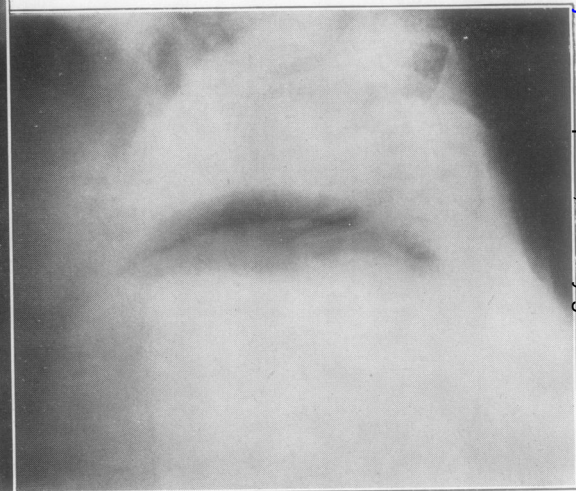
(b)

PLATE X.—(a) Case of bilocular suprahepatic abscess. Postero-anterior film showing (1) slightly raised, well-defined right diaphragm, (2) clear lung field, and (3) two gas spaces with fluid levels below right diaphragm. (b) Postero-anterior film in lateral recumbency of same case showing two gas-containing loculi with a fluid level in each.

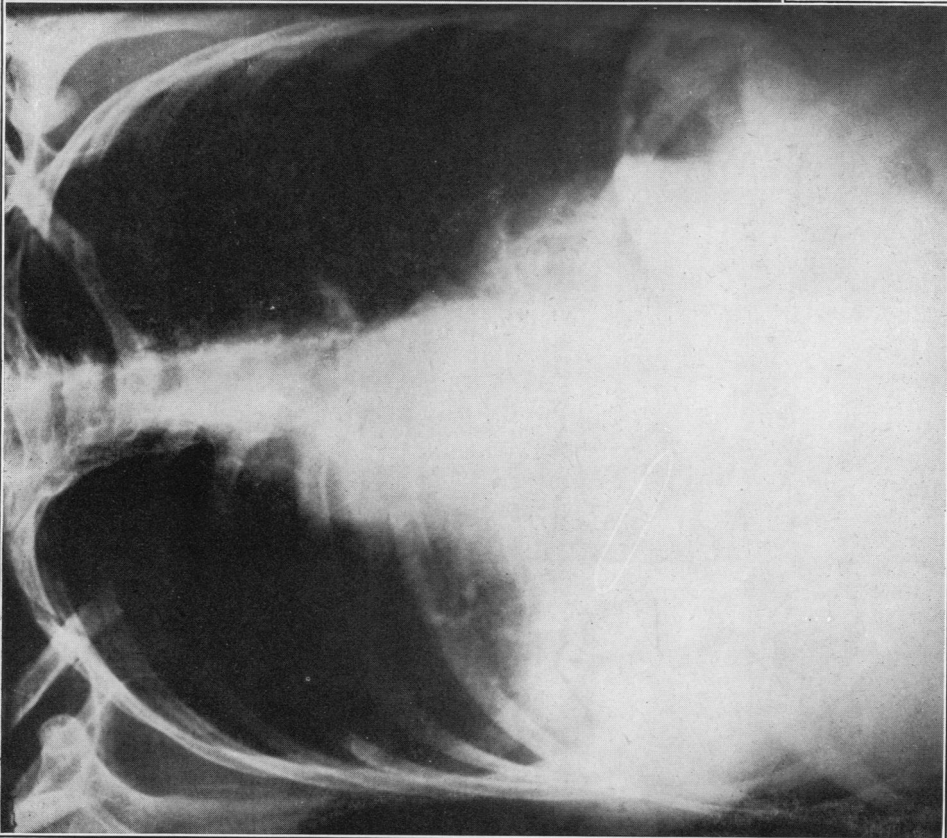


(c)

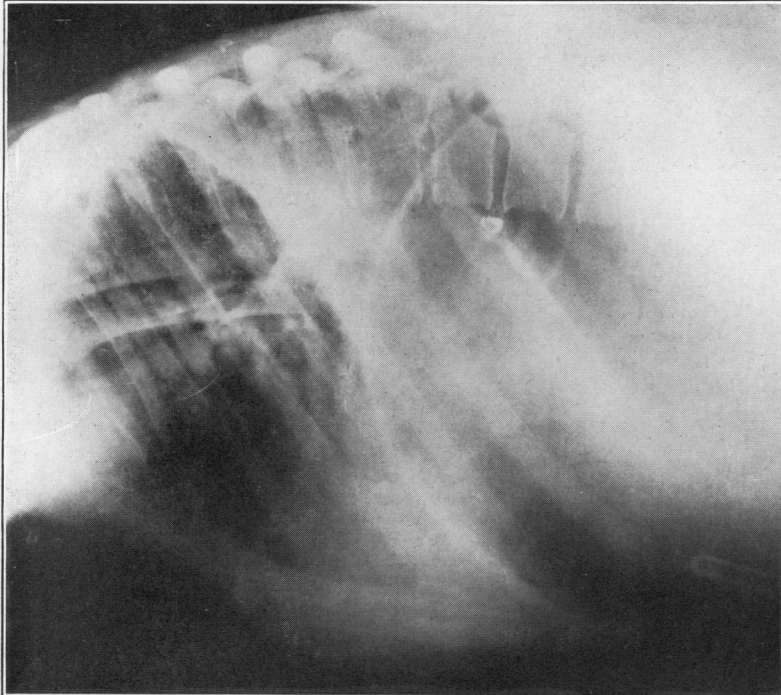
PLATE X.—(c) Case of left suprahepatic abscess. Postero-anterior film showing (1) high left diaphragm, completely obscured by (2) uniform opacity at left base, probably due to fluid, (3) displacement of mediastinum to right, and (4) gas below left diaphragm. No definite fluid level visible. (d) Lateral film of same case showing raised left diaphragm with gas and fluid level beneath it. The right diaphragm is seen crossing the gas space below the left diaphragm.



(d)

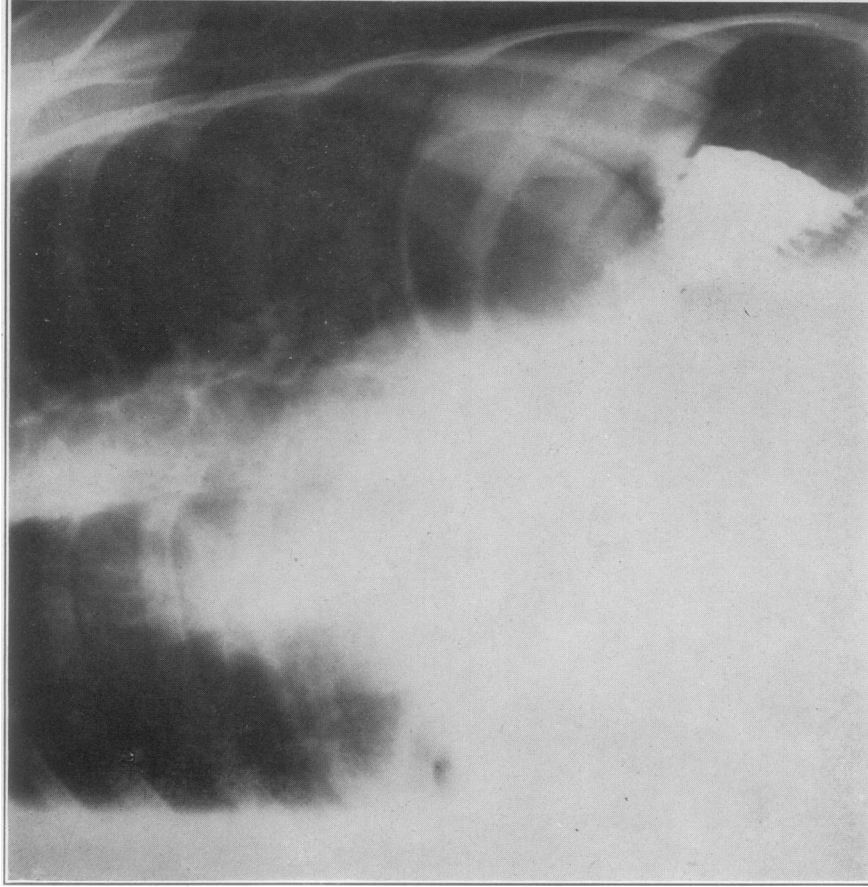


(a)



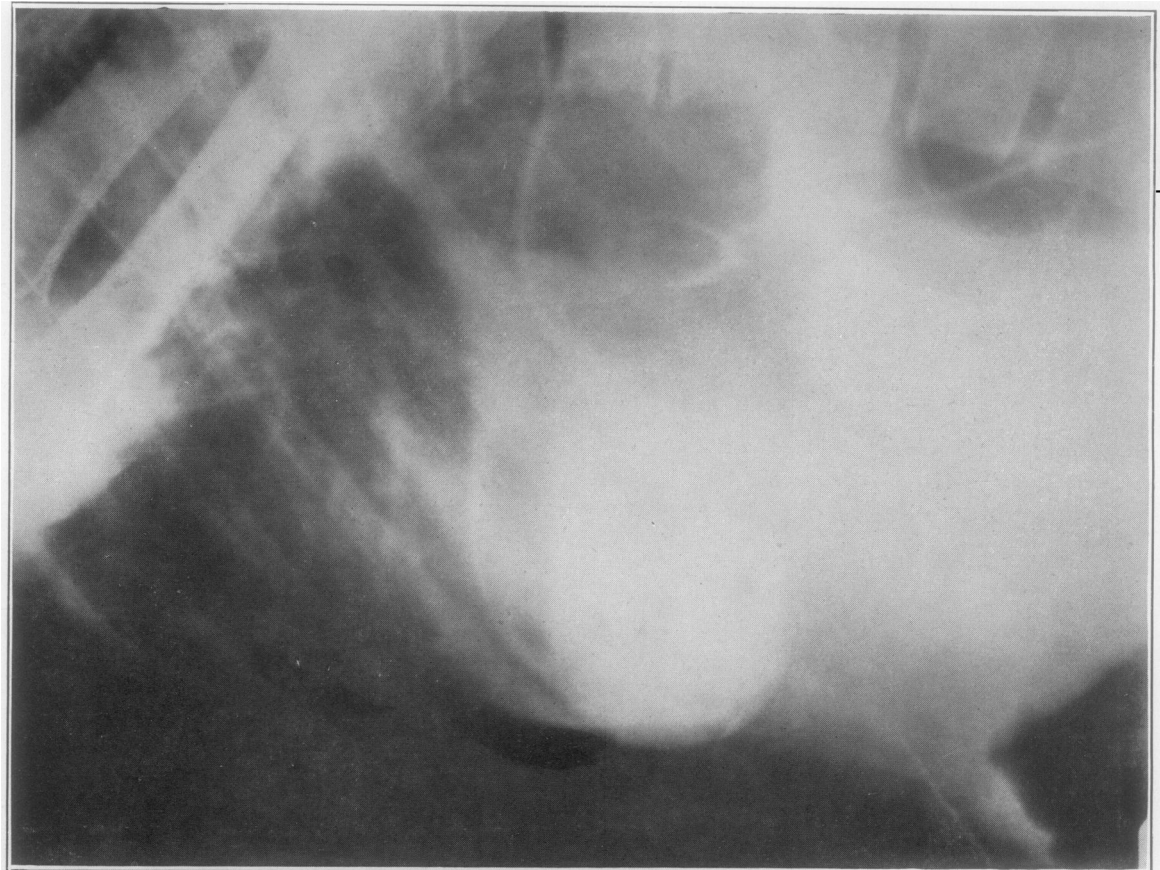
(b)

PLATE XI—(a) Case of bilateral subphrenic abscess (right suprahepatic and left suprahepatic). The right suprahepatic abscess was drained extrapleurally on July 2, 1948, and transpleurally on July 9, 1948. Pyrexia persisted. Postero-anterior film showing (1) drainage tube *in situ* or right side, and (2) high, well defined left diaphragm with gas and fluid level beneath it. The walls of the gas-containing space are irregular. (b) Lateral film of the same case showing (1) high, well-defined left diaphragm, and (2) posterior triangular opacity in lung field.



(a)

PLATE XII.—(a) Oblique film taken after administration of barium meal, showing stomach, (1) spleen, (2) gas under left diaphragm lateral to stomach, (3) gas still present under right diaphragm. (b) Lateral film of same case, taken after administration of barium meal, showing stomach displaced somewhat backwards by (2) left subphrenic abscess containing gas and fluid.



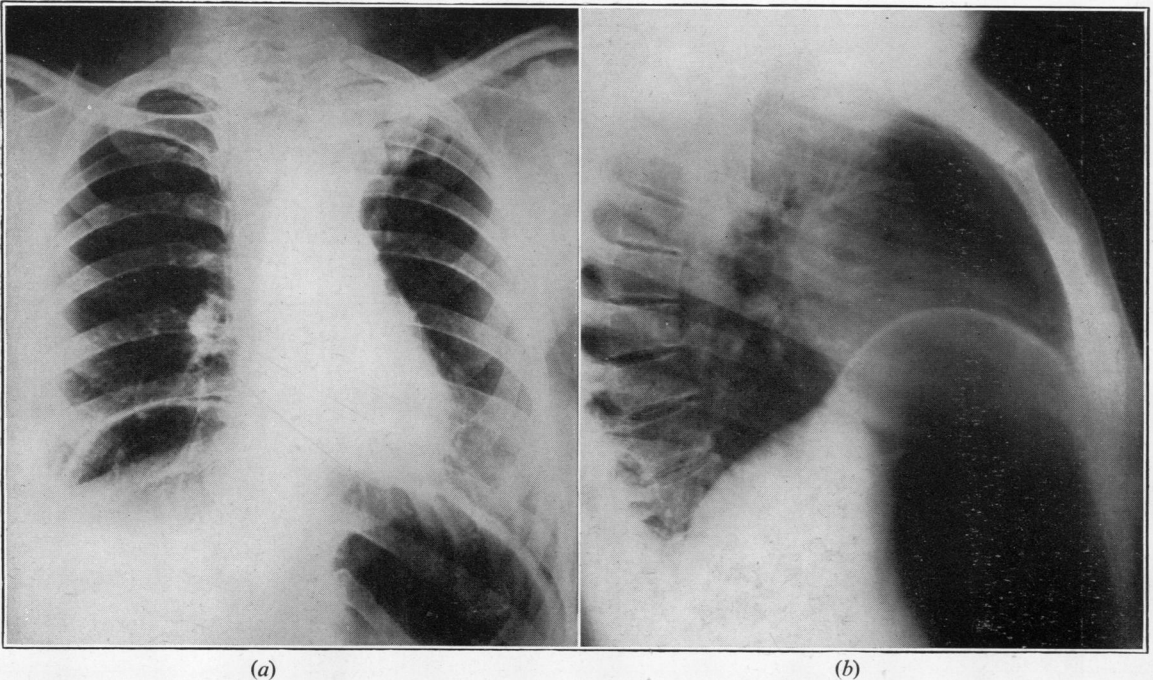


PLATE XIII.—(a) Case of megacolon, showing gas under both domes of diaphragm. The haustrations of the colon are difficult to see, but were more clearly defined in a later film. (b) Lateral film of same case showing large subdiaphragmatic collection of gas in colon.

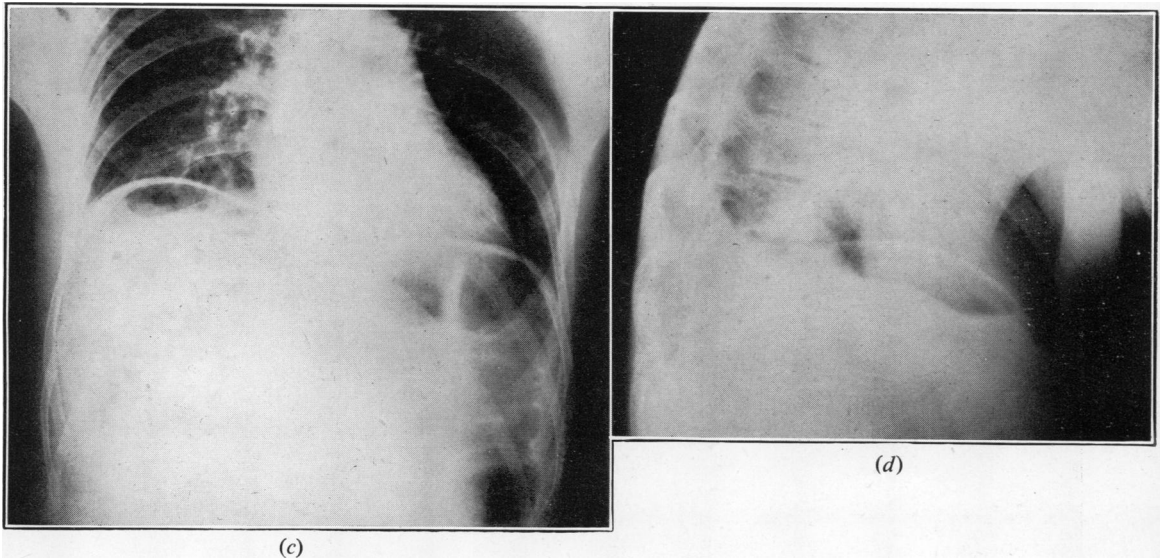


PLATE XIII.—(c) Case of right suprahepatic, gas-containing abscess, in a patient with megacolon. Postero-anterior film showing (1) high, well-defined right diaphragm with underlying gas and fluid level in a subphrenic abscess, and (2) gas in dilated colon beneath left diaphragm. The liver is seen above the fluid level beneath the right diaphragm. (d) Lateral film of same case showing gas-containing abscess cavity below right diaphragm. A fluid level is seen anteriorly. Behind this the liver rises above the level of the fluid.

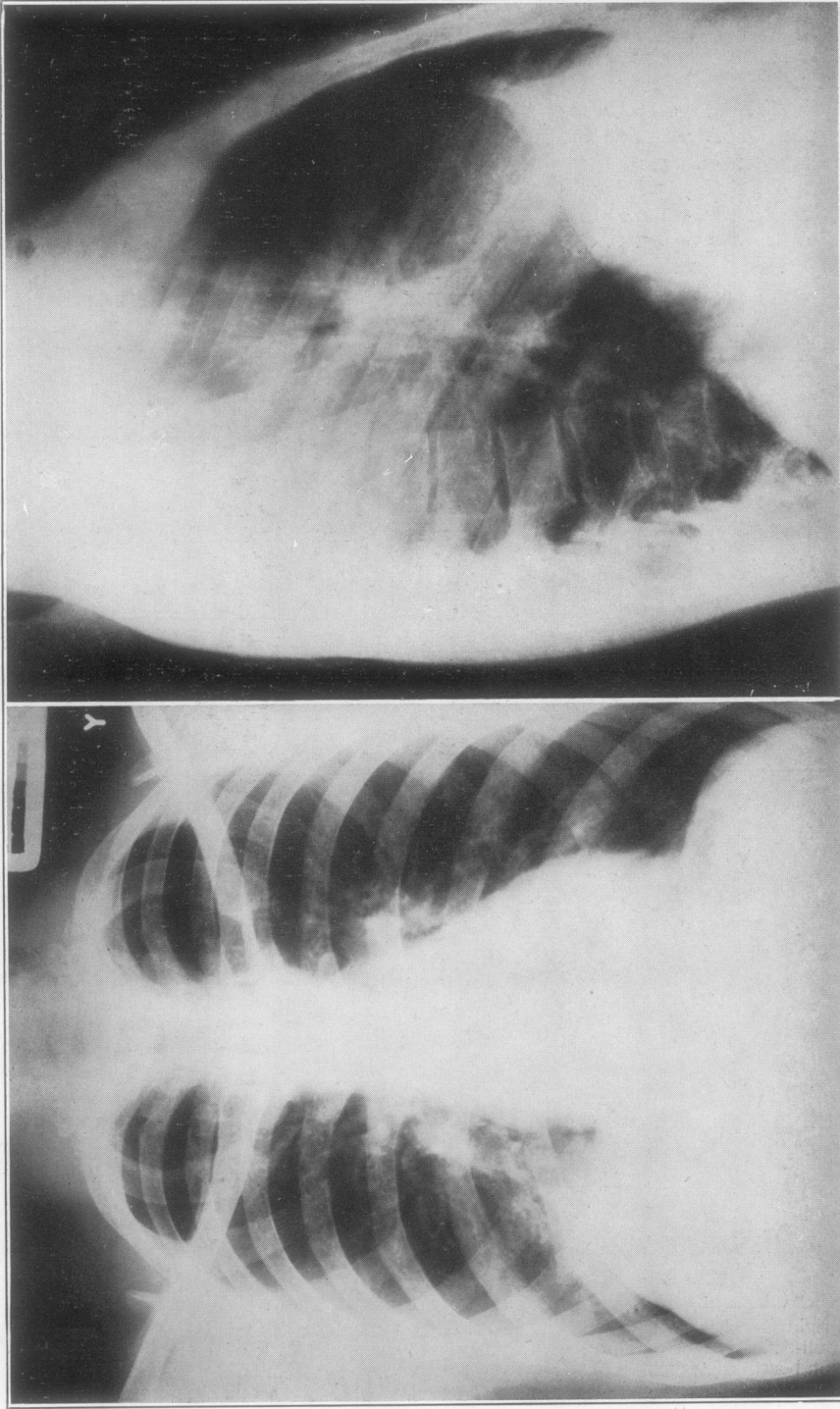


PLATE XIV. Case of chronic right suprahepatic abscess in which diagnostic pneumoperitoneum was performed. Postero-anterior film showing (1) high, well-defined right diaphragm, (2) clear lateral costophrenic angle, and (3) opacity in lung field above highest portion of diaphragm, probably due to pneumonitis. (b) Lateral film of same case showing (1) right diaphragm greatly raised anteriorly, where it is poorly defined, (2) clear posterior costophrenic angle, and (3) opacity in lung field just above highest portion of diaphragm, probably due to pneumonitis.

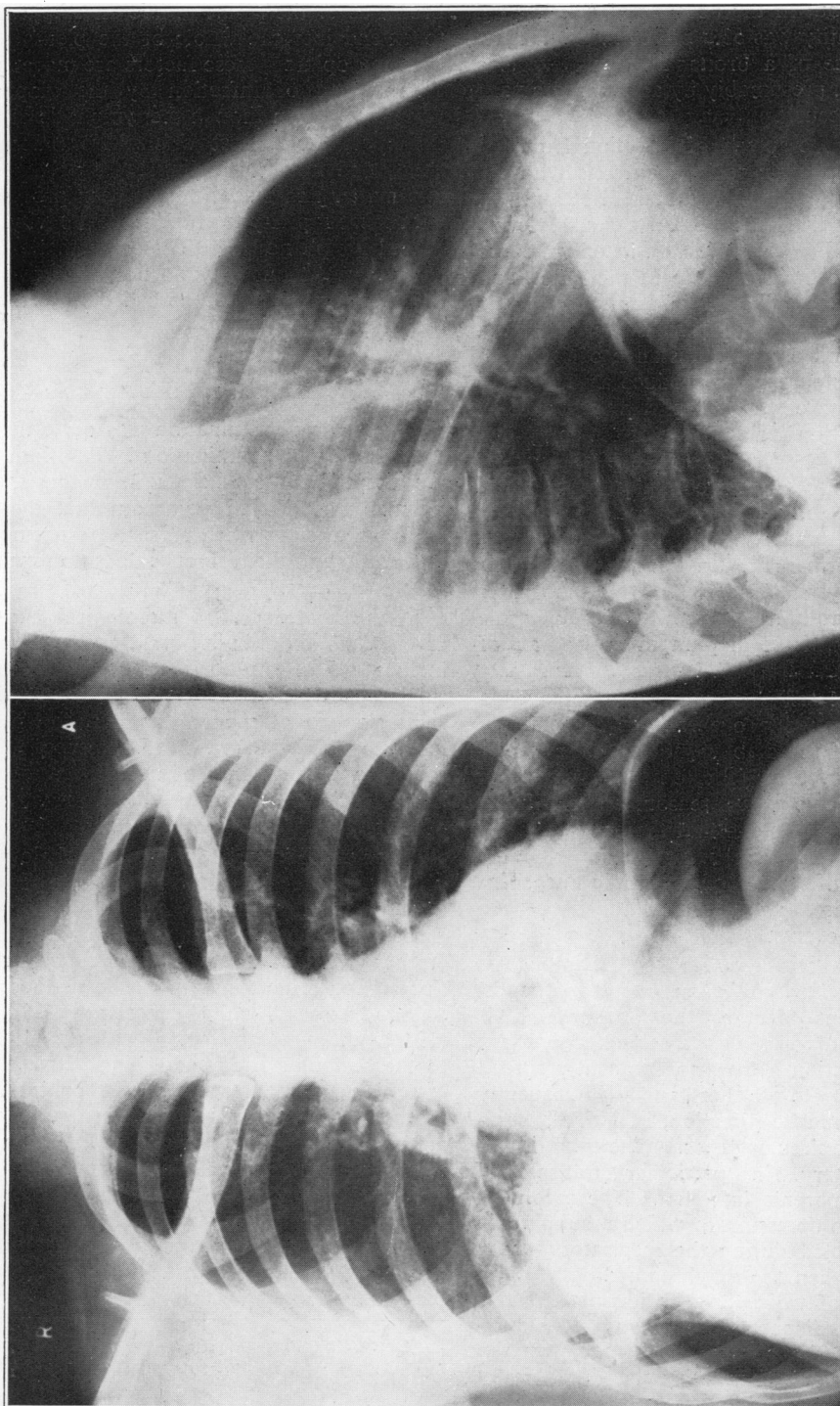


PLATE XV.—(a) Postero-anterior film of same case as in Plate XIV, taken after induction of pneumoperitoneum, showing liver adherent to right diaphragm. (b) Lateral film of the same case, taken after induction of pneumoperitoneum, showing liver adherent to right diaphragm anteriorly.

17.65 per cent, but in the absence of a bronchial fistula the mortality was 53.8 per cent. It appears that the presence of a bronchial fistula exerts a benign influence, presumably by allowing drainage of infection to occur.

The rarity of the spread of infection from the chest to the subphrenic region contrasts with the frequency of spread in the reverse direction. In the large series, comprising 3,533 cases, reviewed by Ochsner and De Bakey (1938), in only 2.5 per cent did the infection originate in the chest, and in their own 75 cases the origin was intrathoracic in only one. In a study of 337 cases of empyema Beye (1932) found only one instance of infection traversing the diaphragm to produce a subphrenic abscess, and in this case the diaphragm was injured during the operation for drainage of the empyema. In the records of the Brompton Hospital the only case of subphrenic abscess complicating thoracic disease which I could find was one reported by Roberts and Nelson (1933), in which right lower lobectomy performed for bronchiectasis was followed by empyema and later by subphrenic abscess. The authors ascribed the subphrenic abscess to injury of the diaphragm at the time of lobectomy.

In the present series only 2 cases followed a thoracic infection; one occurred in consequence of perforation of the diaphragm in a case of pulmonary actinomycosis; the other followed an empyema for no obvious reason. The clinical record of the case suggests that the empyema was the causal lesion, but an empyema may be the first clinical manifestation of a chronic and silent subphrenic abscess, and such may well have been the sequence of events.

Subphrenic abscess so rarely complicates empyema thoracis that when the two conditions are present together the presumption is strongly in favour of the first being the primary one. That infection should so frequently spread from the abdomen to the chest, and so infrequently spread in the opposite direction, is, I think, attributable to two circumstances. The first is ill-considered surgery, in particular transpleural drainage of subphrenic infection. The second is the process of compression to which a subphrenic abscess is subjected during the inspiratory phase of respiration, when the diaphragm descends and in consequence of which an attrition of its fibres occurs which predisposes to its rupture. Downward spread of the abscess is impeded by the abdominal viscera, in particular the liver, or by the formation of limiting peritoneal adhesions. An abscess of the lung usually ruptures into a

bronchus or the pleura, while an empyema is free to extend into the whole of the pleural cavity.

In my opinion, these factors are more important than spread of infection by the diaphragmatic lymphatics, even though the lymphatic drainage in this region is almost wholly from abdomen to chest, and notwithstanding the fact that particulate matter and bacteria have been proved to pass with great rapidity from peritoneal cavity to mediastinal lymph nodes.* These are matters of very great interest, but they do not, I believe, explain the peculiarities of the spread of infection across the diaphragm.

RADIOLOGICAL DIAGNOSIS

Radiological examination was performed in 135 cases and screen examination in 73. Both methods are essential and are helpful in diagnosis, although Lehman and Archer point out that the cardinal radiological signs when present are not necessarily diagnostic and their absence does not exclude subphrenic abscess.

In the present series radiological examination revealed no abnormality in 12 cases (8.9 per cent), including 5 of suprahepatic infection involving a single space. Two of the latter occurred on the right side and three on the left, the percentage incidence being 3.1 and 15.8 respectively. The more frequent absence of radiological changes on the left side as compared with the right is worthy of note.

Other conditions, both above and below the diaphragm, may cause changes similar to those resulting from subphrenic abscess. Elsberg (1901), Barnard (1908), Schwartz (1930), and Lehman and Archer (1937) all state that liver abscess may produce changes indistinguishable from those of subphrenic abscess, while Pancoast (1926), Delario (1934), and Overholt and Donchess (1935) refer to the difficulty in distinguishing between the two conditions. The last three authorities maintain that a large perinephric abscess may give rise to a high diaphragm with some limitation of excursion, but Neuhof and Schlossmann (1942), in a series of 65 cases of perinephric abscess, found only 1 instance in which the diaphragm was raised. This was the case of a child of 9 years old who had a very large abscess, and the elevation of the diaphragm developed late. Radiological

* (Allen, 1936; Bizzozero and Salvioli, 1876; Bolton, 1921; Brown, 1928; Buxton and Torrey, 1906; Cunningham, 1922, 1926; Florey, 1927; Florey and Witts, 1928; Hahn and others, 1944; Higgins and others, 1930; Higgins and Graham, 1929; Higgins and Lemon, 1931; Higgins and Murphy, 1928; Kolossow, 1893; Levi, 1927; MacCallum, 1903; Menville and Ané, 1934; Muscatello, 1895; von Recklinghausen, 1862, 1863.)

examination of the chest was performed in 13 of 50 cases of perinephric abscess studied by the author. In 3 of the 13 patients the diaphragm was raised and showed restricted movement. The radiographs of one of these three patients are shown in Plate IIIc and *d*. Aspiration of the chest withdrew serous fluid from 3 patients, and in two others a radiological diagnosis of pleural effusion was made. Delario (1934) points out that the radiological diagnosis of perinephric abscess may be suggested by such features as obliteration of the outer edges of the psoas shadow, or the demonstration of enlargement of the apparent kidney shadow, stones, or hydronephrosis.

In the first few weeks following an abdominal operation, even when convalescence is uncomplicated, the diaphragm may be elevated and its movement diminished, gas may be present beneath it, and changes may be present in the lung fields. This subject is discussed by Muller and others (1929), Overholt and Donchess (1935), Neuhof and Schlossmann (1942), and Howkins (1948).

Both postero-anterior and lateral films should invariably be taken with the patient in the upright position. Penetrating films are desirable as well as those taken with standard exposures for the chest, for they often demonstrate a diaphragm whose position is obscure in films of normal exposure, and facilitate the recognition of gas in a subphrenic abscess. The patient should be screened whenever he is fit enough for this examination. When necessary, screening can be conducted with the patient in the horizontal position, as an adequate estimate of diaphragmatic movements can be obtained in this posture. The importance of screening has recently been re-emphasized by Thorek (1947), who regards immobility of the diaphragm as much more important than its elevation.

When studying the radiological features attention should be paid to the diaphragm and to the regions above and below it. The changes which may be found are illustrated in Plates IV to X inclusive. The height, mobility, thickness, and definition of the diaphragm must be examined. Normally the diaphragm is freely mobile, thin, and well defined. In many cases of subphrenic abscess it is elevated, its mobility and sharp definition are diminished or lost, and its thickness is increased. The lateral film may demonstrate elevation to be either general or local. A local elevation always overlies the abscess and is a feature of the greatest diagnostic importance.

Elevation and diminished movement are changes in part due to reflex inhibition of the musculature

of the diaphragm, the purpose of which is to rest the inflamed parts. Increase of thickness of the diaphragm, which may be recognized when gas is present beneath it, and loss of definition of its upper border, are changes which are almost certainly due to inflammatory reaction in the diaphragm itself and in the diaphragmatic pleura and the base of the lung. These changes are an expression of acute inflammatory reaction in the tissues surrounding the abscess.

In the region above the diaphragm changes are commonly seen in the lower portions of the lung field. It seems probable that they are caused by a combination of three pathological processes, namely an inflammatory reaction in the lung, similar to that described by Pancoast (1926) in cases of liver abscess, pleural effusion, and collapse of the lower lobes. Pleural effusion, we know, occurs often, while collapse of the lower lobes is not uncommonly found in cases coming to necropsy. A large effusion may completely obscure the diaphragm in the postero-anterior films of normal exposure and so confuse the diagnosis, but the true state of affairs will usually be made obvious by taking lateral and penetrating films. When the diaphragm is high the mechanics of the thorax is altered, so that a pleural effusion may give rise to an atypical radiological shadow.

There is no proof of the occurrence of an inflammatory reaction in the lungs, but in some cases the lateral costophrenic angle remains clear, while patchy shadowing occurs in the region just above the diaphragm. If the abscess can be localized radiologically the shadowing is seen to occur in its immediate neighbourhood (Plate VIIIa and *b* and Plate XIV). These appearances are not those which might be expected of pleural effusion or collapse of the lower lobes, and are, in my opinion, best explained by postulating an inflammatory reaction in the lung similar to that already discussed in the case of the diaphragm. The frequent finding at necropsy of a lower lobe firmly adherent to the diaphragm supports this contention.

Loss of the posterior costophrenic angle in the lateral view is a very constant finding, and a posterior triangular opacity, with its apex above and behind in the paravertebral gutter, is sometimes present. These appearances are usually due to pleural effusion.

In a few cases contralateral displacement of the mediastinum occurs. This is due to elevation of the diaphragm, or to pleural effusion, or to both.

The features to be looked for below the diaphragm are the presence of gas, usually with a

fluid level but occasionally without, and displacement of abdominal viscera. Gas is absent in a majority of cases, and its presence is not essential to the diagnosis of subphrenic abscess. It was present in only 36 cases (26.7 per cent) of the present series. The incidence given by other authors is shown in Table VIII.

TABLE VIII
INCIDENCE OF GAS IN CASES OF SUBPHRENIC ABSCESS

Authority	Number of cases	Percentage with gas
Elsberg (1901)	—	15
Berman (1925)	—	15
Delario (1934)	—	25
Overholt and Donchess (1935)	22	9
Ochsner and De Bakey (1938)	25	30
Faxon (1940)	83	26
Clagett and Tinney (1944) ..	80	25
Present series	135	27

The presence of gas in a subphrenic abscess is easily missed in postero-anterior films of normal exposure for the chest. It is more likely to be seen in the lateral view, but is best demonstrated in the penetrating films. Gas, when present, is valuable in localizing an abscess, and films taken in a variety of positions, as has been pointed out by Sommer (1923), Whipple (1929), and Meller (1930), may afford information regarding its extent.

On the left side it may be difficult to distinguish gas in a subphrenic abscess from gas in the stomach. A point of distinction is that the fluid level in the stomach seldom reaches the lateral body-wall in the postero-anterior film, though it may occasionally do so when the diaphragm is high from any cause or when the stomach is distended. Doubt may be resolved by giving a barium meal and then exposing films in two planes with the patient in slight Trendelenburg position. Plates XIa and b and XIIa and b illustrate the use of a barium meal for this purpose. The lateral view taken after giving the barium distinguishes the gastric air bubble from gas in a left subphrenic abscess. There is a subphrenic abscess on the right side also. The patient was not placed in Trendelenburg's position. This test was first described by Carter (1939) and is helpful even if no gas is present, for it may demonstrate indentation of the stomach or its displacement away from the diaphragm. An alternative procedure, described by Neuhof and Schlossmann (1942), is to distend the stomach with gas by giving the patient a Seidlitz

powder or bicarbonate of soda before submitting him to radiological examination. The differential diagnosis of the causes of gas under the right dome of the diaphragm are discussed by Pendergrass and Kirk (1929) and by Thaxter (1940). Gas in this situation may occupy the peritoneal cavity, the colon, or a dextroposed stomach, as well as a subphrenic abscess. Plate XIIIa and b demonstrates a case of megacolon, and Plate XIIIc and d a case of right subphrenic abscess occurring in a patient with megacolon.

Lilienthal (1937) has advocated inducing a pneumoperitoneum in the diagnosis of subphrenic abscess, and this has also been advised by Wilkie and Clark (1946). In my opinion, this is a dangerous proceeding in the case of an acute abscess because it may rupture adhesions and disseminate infection, but for chronic varieties it is safe and useful. Plates XIV and XV show films, kindly lent by Dr. Blair, of a patient with a chronic abscess, in whom a pneumoperitoneum was induced. They demonstrate the adherence of the liver to the diaphragm.

THE ORIGIN AND CLINICAL SIGNIFICANCE OF GAS IN A SUBPHRENIC ABSCESS

There has been much debate regarding the origin and clinical significance of gas in subphrenic abscess. Neuhof and Schlossmann (1942) discuss three possible sources of gas, namely (1) communication with a hollow viscus, (2) communication with a bronchus, and (3) gas-forming organisms. In all their cases in which gas was present the abscess communicated with a hollow viscus or a bronchus. No gas was found in four cases infected with anaerobic streptococci or *Clostridium welchii*. Wessler and Jaches (1923) state that a subphrenic abscess contains gas only if a communication with the bowel or a bronchus exists, while Delario (1934) and Ochsner and Graves (1933) maintain that gas is found most commonly in these circumstances. Gatewood (1930), on the other hand, postulates a bacterial origin of the gas. Ochsner and Graves claim that gas formation is a late event. Neuhof and Schlossmann assert that the presence of gas is of grave significance because it indicates perforation of a hollow viscus or communication with the bronchial tree. These controversial matters will now be considered.

Of the 36 cases in the present series in which gas was found, the abscess communicated with the bowel, the bronchial tree, or the exterior, as shown in Table IX, in 26 (72.2 per cent). In cases no such communication existed, and in

there was not sufficient evidence on which to base an opinion.

TABLE IX

SOURCE OF GAS IN 26 CASES OF SUBPHRENIC ABSCESS IN WHICH COMMUNICATION WITH A SOURCE OF GAS WAS PRESENT

	Number
Perforation of bowel	19
Bronchial fistula	5
Perforation of bowel and bronchial fistula	1
Penetrating thoraco-abdominal wound ..	1
Total	26

The findings in the 5 cases in which there was no evident communication with a source of gas are as follows: In one case, due to pure *S. aureus* infection, gas was present in the subphrenic abscess 12 days after splenectomy. The only possible origin of gas here appears to be the laparotomy. In the second case gas was present in a subphrenic abscess twenty-three days after cholecystectomy for calculus cholecystitis. In this case *Cl. septicum* occurred as part of a mixed infection and was the probable source of the gas. In the 3 remaining cases anaerobic cultures were not performed. One of these was a "primary" abscess. There was no communication with bowel or bronchus, and no operation was performed prior to the finding of gas. In another case gas was present thirty-four days after appendicectomy and fourteen days after the drainage of an abscess in the right iliac fossa. In the final case gas was found thirty-four days after a penetrating abdominal wound with injury to the liver. No pneumothorax was present at any time. In this case the time interval was too long for the gas to have come from the original injury.

It appears, therefore, that the presence of gas in a subphrenic abscess is usually caused by a communication at some stage with the bowel, or a bronchus, or the exterior. In a few cases there is no such communication, and in these the gas must originate from the infecting organisms or from gas which gains entry to the peritoneal cavity at the time of laparotomy.

Gas is frequently present early in the development of a subphrenic abscess. In 12 of the present cases it was detected within 14 days of onset of the causal lesion or of the operation performed in its treatment. This is in disagreement with the statement of Ochsner and Graves (1933) that gas formation is a late event. It may occur early or

late. In one case gas was first recognized within four days and in another case only after four months.

The mortality of the 36 cases containing gas was 16.6 per cent, whereas the overall mortality for the whole series of 182 cases was 40.1 per cent. This finding does not support the contention of Neuhof and Schlossmann (1942) that the presence of gas is a matter of grave concern.

DIAGNOSTIC ASPIRATION AND EXPLORATORY OPERATION

There is much difference of opinion regarding the value and wisdom of diagnostic aspiration. Controversy is concerned chiefly with the reliability and the safety of the procedure.

Aspiration is an unreliable method of diagnosis. It was used in 58 cases of the present series, and pus was found in the subphrenic abscess in only 14 of these—that is, 24.1 per cent. The findings of other authors are shown in Table X.

TABLE X

NUMBER OF CASES IN WHICH DIAGNOSTIC ASPIRATION DEMONSTRATED PUS IN SUBPHRENIC ABSCESS

Authority	Number aspirated	Pus obtained
Barnard (1908)	18	7 (39%)
Overholt and Donchess (1935)	21	14 (67%)
Neuhof and Schlossmann (1942)	17	14 (82%)
Present series	58*	14 (24%)
Total	114	49 (43%)

* Serous fluid obtained from pleura in 26 other cases.

It is evident from a consideration of the total that pus was withdrawn in only 49 out of 114 cases (43 per cent), and the procedure thus gave negative results more often than not. So high an incidence of failure is a matter of importance, for it is one which may lead to delay in diagnosis and treatment, to the great detriment of the patient. The finding of serous fluid in the pleural cavity is one which is always suggestive of subphrenic infection. Reference to Table X indicates that such fluid was obtained in 26 cases when no pus was found.

Lehman and Archer (1937), Ochsner and De Bakey (1938), Faxon (1940), Claggett and Tinney (1944), and Thorek (1947) believe that, even when it is followed by immediate operation, diagnostic aspiration is unsafe because of the danger of infecting the pleural cavity. Ochsner and De Bakey say, "Diagnostic aspiration should never

be attempted, and in cases in which the diagnosis remains doubtful, exploratory operation should preferably be undertaken."

Barnard (1908), Gatewood (1930), Bogart (1934), Janz (1934), and Szacsavay (1934) maintain that aspiration is safe, but only if performed immediately before operation. Doherty and Rowlands (1931) and Overholt and Donchess (1935) deprecate aspiration, but should it be performed, they advocate the use of the extra-serous route.

Neuhof and Schlossmann (1942) stress the difficulty in diagnosing subphrenic infection on the left side, and conclude that "exploratory aspiration is imperative in all cases with vague clinical and inconclusive features suggesting suppuration in the left subphrenic space." They perform aspiration at the time of operation.

Precise statements regarding the frequency with which infection of the pleural and peritoneal cavities follows aspiration are scanty. In the present series there was evidence that infection of the pleura occurred in consequence of aspiration in only one of the 58 cases in which it was performed. Lehman and Archer (1937) report two cases. Empyema followed aspiration in one of the 21 cases recorded by Overholt and Donchess (1935). In one of Barnard's (1908) cases no pus was obtained at aspiration, but three hours later the patient died and at necropsy it was found that one and a half pints of turbid offensive pus had passed through the puncture track from the right subphrenic space into the pleura, and that the lung was collapsed. In none of Neuhof and Schlossmann's (1942) cases, in all of which the abscess was situated on the left side, did pleural infection ensue. Aspiration was responsible for pleural infection in 3 of the 114 cases represented in Table X.

From these observations it is evident that the pleura may be infected in consequence of diagnostic aspiration, whether pus is withdrawn or not, but in only a small percentage of cases does this event occur.

Aspiration is a method of diagnosis which, in my opinion, should not be employed, chiefly because it is unreliable but partly because of the risk of pleural infection which it entails, though this risk is admittedly small. If aspiration fails, exploratory operation will alone exclude the diagnosis; on the other hand, if pus is withdrawn an operation will be required for the drainage of the abscess.

In those cases in which a study of the clinical and radiological findings leaves the diagnosis in doubt, the wisest course is to perform an explora-

tory operation under local anaesthesia by the extra-serous route, if necessary both from in front and from behind.

THE EFFECT OF DRAINAGE UPON MORTALITY

The mortality for the whole series of 182 cases was 40.1 per cent; 139 abscesses were drained with a mortality of 26.6 per cent; the mortality of those which were not subjected to operation was 83 per cent.

Barnard stated in 1908 that drainage was more safely performed from behind than from in front. The mortality in this series, when drainage was performed from behind, from the side, and from in front, is shown in Table XI. The figures support Barnard's contention.

TABLE XI
INFLUENCE ON MORTALITY OF SITE OF DRAINAGE OF
SUBPHRENIC INFECTION

Site of drainage	Number drained	Died
Posterior	48	6 (13%)
Lateral	20	4 (20%)
Anterior	60	22 (37%)

Table XII illustrates the very striking difference in mortality of drainage by the trans-serous and extra-serous routes, and the difference is one which merits special emphasis. By trans-serous drainage is meant drainage across the pleural cavity, whether the pleura be free or adherent, or drainage across the free peritoneal cavity.

Another feature which is worthy of note is that trans-serous drainage was performed twice as often as drainage by the extra-serous method, despite the fact that many of the cases were treated during the last decade.

TABLE XII
INFLUENCE ON MORTALITY OF TRANS-SEROUS AND EXTRA-SEROUS DRAINAGE OF SUBPHRENIC INFECTION

Method of drainage	Number drained	Died
Extra-serous ..	42	4 (11%)
Trans-serous ..	83	27 (33%)

The great importance of the bearing of the method of drainage on mortality was clearly demonstrated in 1933 by Ochsner and Graves, and again in 1938 by Ochsner and De Bakey. The figures given in the collective review of the latter authors are as follows: in 394 cases treated by

trans-serous drainage the mortality was 36.2 per cent, whereas in 211 cases treated by the extra-serous method the mortality was 20.8 per cent; in 37 cases drained extra-serously by Ochsner and De Bakey themselves the mortality was only 10.8 per cent. The importance of the extra-serous route has been stressed more recently by Clagett and Tinney (1944), Zaveleta (1945), Lewenstein (1946), and Thorek (1947).

That the proportion of cases treated by trans-serous drainage in the present series is so high is evidence that the superiority of the extra-serous method is even yet not fully appreciated. It was possible to determine the method of drainage used in 74 patients treated since the beginning of 1939. In 60 per cent of these the trans-serous method was employed. Prior to 1939 the trans-serous route was utilized in 74.5 per cent of cases.

A matter which also requires emphasis is that adherence of the pleura is no guarantee against the spread of infection after trans-pleural drainage. This has been stressed by Doherty and Rowlands (1931). In the present series there was one death amongst the four patients on whom drainage was performed through a pleural cavity which was stated to have been obliterated by adhesions. Barnard (1908) recorded 5 cases in which pleural adhesions gave way when the diaphragm descended after the abscess had been drained. This happened twice at operation and three times a day or two later.

The extra-serous method of drainage should, I believe, invariably be adopted in the treatment of subphrenic abscess, even when the serosa is adherent. By its employment the mortality of operation should be reduced to the neighbourhood of 10 per cent.

The entire subphrenic region can be reached by the posterior and anterior extra-serous routes described by Nather and Ochsner (1923) and by Clairmont and Meyer (1926). Which of these routes be chosen will depend upon the location of the abscess. If it is equally accessible from behind or in front then the posterior route should be selected.

SUMMARY

1. A series of 182 cases of subphrenic abscess is reviewed.
2. Only one intraperitoneal subphrenic space is recognized above the right lobe of the liver.
3. Marked clinical differences were found between suprahepatic and infrahepatic infection

on the right side. On the left side these differences were less striking.

4. Infection occurred on the left side in fifty cases. It presented certain clinical peculiarities, and was attended by a high mortality.

5. A brief account of multiple-space infection is given and the high mortality noted, especially when the abscess is bilateral.

6. Primary subphrenic abscess occurred in seventeen cases, and was attended by a relatively low mortality.

7. Chronic subphrenic abscess is defined, and sixteen cases of the condition are discussed.

8. A majority of the cases of subphrenic abscess secondary to intra-abdominal inflammation followed neighbouring infection. Many of those in which the inflammation was more remote from the subphrenic region were residual abscesses following general peritonitis. No evidence was obtained to suggest that spread of infection occurred via the abdominal lymphatic vessels.

9. Nineteen cases of subphrenic abscess in association with liver abscess are presented, and the high mortality is noted.

10. Serous pleural effusion occurred in about 25 per cent of cases. The cause of the effusion was seldom in the chest, and the fluid rarely became purulent.

11. Intrathoracic suppuration occurred in slightly more than 25 per cent of cases, and was usually due to some such cause as perforation of the diaphragm or transpleural drainage of the subphrenic abscess. No evidence was obtained to suggest that infection commonly spreads from the subphrenic region to the chest via the lymphatic pathways.

12. When a bronchial fistula was present in addition to intrathoracic suppuration, the mortality was significantly diminished.

13. The radiological diagnosis is discussed in detail and certain difficulties are considered.

14. Gas was demonstrated in the abscess in 26.7 per cent of cases examined radiologically. In most cases it was caused by communication between the abscess and the bowel, the bronchial tree, or the exterior. The mortality of the cases in which gas was present was lower than average for the series.

15. Diagnostic aspiration is not advised because of its unreliability, and also because of the danger of infecting the pleura. This danger is less than might be expected.

16. Trans-serous drainage is attended by a much higher mortality than extra-serous drainage and is therefore condemned.

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