SUBPHRENIC ABSCESS, WITH PARTICULAR REFERENCE TO THE SPREAD OF INFECTION

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H. R. S. Harley, M.S., F.R.C.S.

Department of Thoracic Surgery, The United Cardiff Hospitals, and Sully Hospital

IT IS MY purpose to determine how infection reaches the subphrenic spaces, and also how it spreads from them, across the diaphragm, to the chest. I can find no reference to this subject in the writings of John Hunter, but we know that he examined the subphrenic spaces, for, at an autopsy examination performed by him he found an appendix caeci " which was vastly inflamed, ulcerated and in some places mortified," and he described the adhesions which were present between the diaphragm and its subjacent viscera. His comments on this case indicate that he was familiar with inflammation of the appendix, and, with rare vision, he recognised that the features of intestinal obstruction, which had been present during life, must have been due to paralysis of the bowel, because no mechanical obstruction was found. The great interest which Hunter displayed in the subject of lymphatic absorption, and which he shared with his brother William, would, I hope, have made the subject of this lecture acceptable to him. Amongst his many wise and thoughtful words John Hunter said: " To be puzzled is the first step to knowledge." These words must surely encourage us to further effort when the solution of our problems appears to be beyond our reach.

My observations are based upon a study of the clinical and post-mortem material concerning 188 patients suffering from subphrenic abscesses, most of whom were admitted to either voluntary teaching or special chest hospitals in this country. To all those who have provided this material my grateful thanks are due.

Some aspects of the aetiology of subphrenic infection are pertinent to the present subject. Table I shows that 18 (9.6 per cent.) of the abscesses studied were primary, by which I mean that no definite cause was found to account for them, and that 170 abscesses were secondary to some other condition.

TABLE I

INCIDENCE OF PRIMARY AND SECONDARY SUBPHRENIC ABSCESSES

Type of abscess	Number	Per cent.
Primary	18	9.6
Secondary	170	90.4
TOTAL	188	100.0

With regard to the method of spread of infection within the abdomen the secondary abscesses are important. Table II enumerates the sources

of infection in the 170 patients with this type of abscess, and shows that about 90 per cent. of them were caused by intra-abdominal infections or operations.

TABLE II
SOURCE OF INFECTION IN 170 SECONDARY SUBPHRENIC ABSCESSES

Source of Infection	Number of Cases	Per cent.
Intra-abdominal infection or operation	154	90.6
Penetrating wounds of abdomen	10	5.9
Haematogenous infection	4	2.3
Intrathoracic infection	2	1.2
TOTAL	170	100.0

The numbers of intraperitoneal and extraperitoneal abscesses are shown in Table III. None of the extraperitoneal abscesses were primary,

TABLE III
INCIDENCE OF INTRAPERITONEAL, EXTRAPERITONEAL AND COMBINED ABSCESSES

Type of Abscess	Number	Per cent.
Intraperitoneal	171	90.5
Extraperitoneal	14	7.9
Combined	2	1.1
Not known	1	0.5
TOTAL	188	100.0

and when the aetiology of the two varieties of abscess were compared striking differences were apparent. The great majority of the intraperitoneal abscesses occurred in consequence of intraperitoneal inflammation, the three most important causes of which were perforated peptic ulcer (33.3 per cent.), acute appendicitis (20.5 per cent.), and abdominal operations (16.4 per cent.). On the other hand, intraperitoneal inflammation was a rare cause of extraperitoneal abscesses, which usually followed infection of the adjacent extraperitoneal tissues caused by blood-borne infection, penetrating wounds, abdominal operations, or localised inflammatory conditions, such as perinephric abscess, liver abscess, or osteomyelitis of a lumber vertebra. Not a single extraperitoneal abscess was caused by perforation of a peptic ulcer, and only one was due to acute appendicitis. Unfortunately there was no information about the position or pathological condition of the appendix in this case. On the other hand, three of the four haematogenous abscesses were extraperitoneal in location, so that blood-borne infection accounted for 21.4 per cent. of extraperitoneal abscesses, as compared with only 0.6 per cent. of intraperitoneal ones.

It may be stated that almost all secondary intraperitoneal abscesses occur in consequence of intraperitoneal inflammation, and that almost all extraperitoneal ones are caused by infection of the adjacent extra-

peritoneal tissues by blood-borne organisms, or as the result of local damage or infection. This strongly suggests that infection spreads from distant parts of the peritoneal cavity to the subphrenic spaces by an intraperitoneal route.

The way in which infection spreads in the abdomen to reach the subphrenic spaces

As shown in Table II above 154 abscesses were secondary to an intra-abdominal infection or operation, and Table IV illustrates my conception of how infection reached the subphrenic spaces in these patients.

TABLE IV

METHODS OF SPREAD OF INFECTION TO THE SUBPHRENIC SPACES IN 154 ABSCESSES SECONDARY TO INTRA-ABDOMINAL INFLAMMATION

Method of Spread of Infection	Number of Cases	Per cent.
Direct spread from neighbouring focus	100	64.9
Residual abscess following general peritonitis	15	9.7
Intraperitoneal spread via paracolic gutter	4	2.6
Via portal vein (suppurative pylephlebitis)	4	2.6
Not determined	31	20.1
TOTAL	154	99.9

In 100 patients the abscess was caused by an inflammatory process in its neighbourhood. Ninety of these abscesses were intraperitoneal and 10 of them were extraperitoneal, and infection spread by direct continuity, either within the peritoneal cavity, or in the extraperitoneal tissues. In the remaining 54 patients, all except one of whom had an intraperitoneal abscess, the subphrenic spaces were infected from distant parts of the abdomen, chiefly from the appendix, and it is necessary to ascertain how the infection travelled.

Barnard (1908) stated that appendicitis may affect the subphrenic spaces in four ways, namely (1) as part of a diffuse peritonitis ; (2) by a more or less slow and direct extension up the peritoneal fossae from the pelvis ; (3) through the medium of the portal vein, causing pylephlebitis ; and (4) by lymphatic extension, either up the right retroperitoneal cellular tissues, or up the lymphatics accompanying the deep epigastric artery to the falciform ligament. There are thus three possible routes of spread from distant parts of the abdomen, intraperitoneal, vascular and lymphatic.

There has been much controversy regarding the frequency of these methods of spread. Munro (1905) stressed the importance of retroperitoneal lymphatics and the portal vein, though he also recognised the occurrence of intraperitoneal spread. He said " that infection of the lymphatic channels should take place with great frequency in inflammation of the appendix need cause no surprise, when we consider that that organ and the adjacent walls of the caecum are peculiarly rich in lymphoid tissue. Clinically, we see, almost universally, some evidence of absorption

in the mesenteric nodes. This is notably evident in children, and it frequently happens that a bunch or string of tender nodes is much more in evidence on palpation of the unopened abdomen than the appendix itself, in which, indeed, the gross infective process may have subsided." Munro goes on to say, " an analogous condition is seen in the cervical lymph nodes during and subsequent to microbic invasions of the pharyngeal group of lymphatic organs. It is on a similar basis that we should look for more frequent evidences of lymphatic invasions along the retroperitoneal lumbar chain of lymphatics, presupposing that there is a liberal communication between the lymphatics of the caeco-appendix walls and this lymphatic chain." Lockwood (1921) believed this to be the main route of the current from the appendix and caecum.

Munro (1905) maintained that lymphatic pathways extend along the retroperitoneal tissues to the liver, diaphragm and chest, and that the lymphatic vessels of the diaphragm communicate with those of the peritoneum. Spread via the retroperitoneal lymphatics was also favoured by Truesdale (1933), Janz (1934) and Lehman and Archer (1937) who believed that this is the common method of infection of the subphrenic spaces in the absence of general peritonitis. According to Truesdale, the lymph from the appendix drains into the appendicular and ileocaecal nodes and thence into vessels which accompany the superior mesenteric and portal veins to the liver. After pursuing " a most intricate course " the lymphatic vessels finally reach the diaphragm where infection localises to form a subphrenic abscess.

On the other hand, Overholt and Donchess (1935), Ochsner and DeBakey (1938), Carter (1939), Faxon (1940), Spalding (1943) and Thorek (1947) all regard spread from distant parts of the abdomen as occurring intraperitoneally. Neuhof and Schlossmann (1942) found evidence of lymphatic spread in only two out of 33 cases of subphrenic abscess on the left side.

As stated above, infection spread to the subphrenic spaces from distant parts of the abdomen in 54 patients. In every case except one the abscess was an intraperitoneal one, and this strongly suggests that infection travelled via the peritoneal cavity. In 19 of the patients there was good evidence in favour of intraperitoneal spread, for in 15 of them the abscess was a residual one following general peritonitis, while in four there was operative or necropsy evidence of inflammation of the peritoneum of the paracolic gutter. In four patients infection travelled via the portal vein causing suppurative pylephlebitis, and subsequent to this a subphrenic abscess. In the case of 23 patients, therefore, the method of spread of infection was clear, and was via either the peritoneal cavity or the portal vein, but in the remaining 31 patients the route of spread was not obvious. It is most unlikely that spread occurred via the portal vein in these cases, because the patients would almost certainly have died and the evidence would have been obtained at necropsy.

In none of these 31 patients was there any evidence that spread of infection via lymphatic pathways had occurred. The lymphatic vessels draining the appendix accompany the ileocolic and superior mesenteric arteries, according to Gray's Anatomy, and not the veins, as postulated by Truesdale (1933). They pass through appendicular, ileocaecal and superior mesenteric nodes to reach the intestinal lymphatic trunks, and thence the cysterna chyli. Lymph could reach the liver only by retrograde flow from the region of the superior mesenteric and coeliac nodes, and thence via the hepatic nodes. Infection of the liver by such a devious route seems most unlikely. Furthermore, even if the liver were infected in this way it is difficult to imagine that the organisms would be carried to the region beneath the diaphragm, there to form a localised abscess within the peritoneal cavity.

The lymphatic vessels of the liver communicate with three groups of nodes in relationship with the diaphragm, namely, the anterior diaphragmatic nodes, situated behind the base of the xiphoid process, the right lateral diaphragmatic nodes, which surround the terminal part of the inferior vena cava, and the paracardial nodes, which surround the cardiac orifice of the stomach. The anterior and right lateral groups of nodes are placed on the thoracic aspect of the diaphragm. If infection did spread from the liver to these three groups of nodes abscesses should occur in relationship to them, but this is not the case. Furthermore, infection of the anterior and right lateral groups of nodes should occasion an empyema thoracis rather than a subphrenic abscess.

If infection from the appendix commonly spreads along retroperitoneal lymphatic pathways appendicitis should be a common cause of extraperitoneal subphrenic abscesses, yet of 36 abscesses secondary to appendicitis 35 were intraperitoneal, and only one was extraperitoneal. All other extraperitoneal abscesses studied were due to either local or blood stream infection.

The evidence against lymphatic spread of infection from distant parts of the abdomen may now be summarised. (1) All the distant abdominal conditions responsible for subphrenic abscesses were calculated to contaminate the peritoneal cavity, and all except one of the resulting subphrenic abscesses were intraperitoneal in location. (2) No evidence for lymphatic spread was found. (3) No direct communications between the appendicular and diaphragmatic lymph nodes exist. (4) Subphrenic abscesses are not related topographically to lymph nodes. (5) Two of the three groups of diaphragmatic lymph nodes, including those on the right side, are situated on the thoracic aspect of the diaphragm.

It remains now to give the evidence in favour of intraperitoneal spread of infection from distant parts of the abdomen to the subphrenic region in the 31 patients in whom no evidence was found to explain how the infection had travelled. In 30 of the patients the subphrenic abscesses were intraperitoneal, and the lesions responsible for them were all liable

to contaminate the peritoneal cavity. Much the commonest of these lesions was acute appendicitis, others included surgical induction of labour, diverticulitis, pyosalpinx and volvulus. As stated earlier almost all intraperitoneal abscesses are caused by conditions which contaminate the peritoneal cavity, and almost all abscesses caused by distant intra-abdominal infection are of the intraperitoneal variety. These facts, taken in conjunction with the evidence adduced against lymphatic spread, makes it nearly certain that, in the cases under consideration, infection spread within the peritoneal cavity. Furthermore, whenever there was evidence as to how infection had spread the intraperitoneal route was the one followed, except in the few cases when infection travelled via the portal vein.

Granted that infection does spread within the peritoneal cavity it is yet to be determined how it does so. I believe that infection is disseminated by the movement of infected peritoneal fluid poured out from the site of intraperitoneal inflammation. When general peritonitis occurs this process is easy to understand, but difficulty arises when general peritonitis does not occur.

Lehman and Archer (1937) attacked this method of spread of infection for three reasons: (1) There is often no evidence of peritonitis in the region between the causal infection and the subphrenic abscess. (2) After peritoneal inflammation is established there is little opportunity for fluids to flow. Lehman and Archer state "The gross pathologic picture presented by any extensive suppurative peritonitis bears out this contention. Pockets of pus are found in all portions of the peritoneum, even completely anterior against the anterior abdominal wall." (3) Infection is frequently located above the liver without involvement of the right infrahepatic space, the low spot topographically in the upper abdomen when the patient is recumbent.

Lehman and Archer's arguments can be answered. The passage of fluid need cause no inflammation in the region between the causal infection and the resulting abscess. As Ochsner and DeBakey (1938) pointed out after peritoneal infection occurs there is an exudative stage during which there is an outpouring of bacteria-laden fluid, which can flow freely before limiting adhesions occur. The frequency of involvement of the suprahepatic as compared with the infrahepatic spaces is probably due to local and mechanical factors. Infected fluid is conveyed to the suprahepatic region by the force of gravity, or because of hydrostatic pressure gradients discussed below, and infection localizes there partly because the suprahepatic region approaches a closed space, and partly because of the continuous movement occurring between the diaphragm and its related viscera.

It is easy to demonstrate by screen examination that pleural fluids move rapidly under the influence of gravity, and there is no reason why peritoneal fluids should not do so before adhesions form. Mitchell

(1940) studied the flow of fine barium emulsion injected into the peritoneal cavity of still-born infants and showed that it was governed by the same physical laws which regulate the flow of fluids elsewhere. Thus, the barium emulsion ran down slopes, and accumulated in depressions where it tended to be retained by natural barriers.

In the recumbent position, which is usually assumed after an adbominal emergency, or during sleep, the region where the diaphragm joins the posterior abdominal wall forms a low zone to which fluid will gravitate, and from where it will be spread between the diaphragm and its related viscera by the movement occurring between them. This action can be shown by placing a moistened cover slip on a glass slide. If a drop of dye is placed near the cover slip, and the latter is then moved to and fro, the dye soon spreads between the surfaces of the cover slip and the slide. This forms a reasonable hypothesis to explain how infection can spread from the lower abdomen to the subphrenic spaces.

There is also another mechanism whereby infected peritoneal fluid may be conveyed to the subphrenic spaces from elsewhere in the abdomen. Overholt (1931) showed that in the horizontal dog the hydrostatic pressure in the suprahepatic region is lower than it is elsewhere in the abdomen, and that the pressure varies with the phases of respiration. When respiration is quiet the suprahepatic pressure falls during inspiration and rises during expiration. These respiratory variations have been confirmed by Overholt and Donchess (1935), and they were able to demonstrate similar changes in two patients undergoing drainage for subphrenic abscess. Banyai (1946) recorded the same events in patients undergoing pneumoperitonium treatment for tuberculosis.

Overholt (1931) noticed that if the dog strains, and develops an abdominal type of respiration the respiratory variations are reversed. Riviere, and most other workers prior to Overholt, found that the intraperitoneal pressure varied inversely with the intrapleural pressure, but Livingston (1932) showed that this depended upon the type of respiration. When breathing was normal or thoracic the intraperitoneal pressure fell during inspiration, but when it was abdominal in type the pressure rose during inspiration. During inspiration elevation of the costal arches and reflex relaxation of the abdominal wall tend to increase the abdominal capacity and lower the intra-abdominal pressure, whilst descent of the diaphragm has the opposite effect. Once this is appreciated the apparently conflicting statements of different workers become readily understandable.

Overholt (1931) also showed for the dog that the hydrostatic pressures in various parts of the peritoneal cavity were dependent upon posture, being greater in dependent than in other areas. This was confirmed in the experimental animal by Lam (1939) and by Rushmer (1946), and for man, after laparotomy had been performed, by Drye (1948). Drye showed that when an individual is supine the pressure is the same in all regions of the peritoneal cavity, but that when he is upright the pressure

is three times as great in the lower as in the upper abdomen. The average general pressure in the supine position was 8 cms. of water. In the erect position the pressures recorded in the upper and lower abdomen were respectively 7 cms. and 19 cms. of water. These experiments confirm the belief of Wildesgans (1923), of Krause (1927) and of Hitzenberger (1929) that the abdomen behaves mechanically like a closed box with partially rigid and partially flexible walls, and that it obeys the appropriate hydrostatic laws.

It seems probable, as suggested by Overholt and Donchess (1935), that the hydrostatic pressure differences which develop between the lower and upper abdomen in the erect posture are capable of conveying infected peritoneal fluid from the lower abdomen to the subphrenic region. If this be true the position of nursing is unlikely to affect the development of subphrenic infection.

Spalding (1943) postulated that when a patient is in Fowler's position the presence of a suprahepatic pneumoperitoneum at low pressure plays an important part by aspirating infected fluid in bulk, together with contained particles of macroscopic size, from the infrahepatic to the suprahepatic region. He pointed out the frequent association of subphrenic abscess with conditions causing pneumoperitoneum. I do not believe that pneumoperitoneum acts in this way, but it may well facilitate the spread of infection to the subphrenic spaces by opening them up.

A third possible force which might convey peritoneal fluid to the subphrenic region is that of capillarity. I do not believe that this force is significant in this respect, and furthermore, it could act only when there is a surface of separation between liquid and gas, or between two liquids of different constitution.

It thus appears that two forces are available which can cause infected peritoneal fluid to move about the peritoneal cavity, and to reach the subphrenic region, namely, gravity, which will operate in the recumbent position, and hydrostatic pressure, which may operate in the upright position.

The relationship between liver and subphrenic abscesses

The association of liver and subphrenic abscesses is important for it occurred in 21 (11.2 per cent.) out of the 188 patients, and the aetiological relationship of the two conditions is of interest. The patients were divided into three groups, namely :—

(1) Patients in whom the liver abscess was apparently the primary disease within the abdomen, and in whom the subphrenic abscess occurred in consequence of it by direct spread of infection (Fig. 1).

(2) Patients in whom the subphrenic abscess was apparently a primary one, and in whom the liver abscess occurred in consequence of it by direct spread of infection (Fig. 2).

Fig. 1. Primary liver abscess causing secondary subphrenic abscess by direct spread of infection.

(3) Patients in whom there was some condition elsewhere in the abdomen, such as appendicitis, or perforation of a peptic ulcer, which preceded infection of both the liver and the subphrenic spaces.

There were two patients each in groups (1) and (2), and 17 in group (3). In all four patients of the first two groups the liver and subphrenic abscesses were in direct communication with each other. Although the clinical records of the two patients classified as group (2) suggested that the subphrenic abscesses preceded that in the liver, a study of the cases in group (3) indicates that infection readily spreads from the liver to the subphrenic spaces, but not in the reverse direction. It is, therefore, probable that these two patients harboured an unsuspected primary liver abscess, to which the subphrenic abscess was in reality secondary, and that they should have been classified in group (1).

It was difficult to be certain of the aetiological relationships of the liver and subphrenic abscesses in the 17 patients of group (3), but three sub-groups were separated, as follows :—

(1) Patients in whom the subphrenic and hepatic abscesses were both secondary to some other abdominal condition, but in whom they developed independently of each other. Ten cases (Fig. 3).

(2) Patients in whom the liver abscess was secondary to some other abdominal condition, and in whom infection then spread from the liver to the subphrenic spaces. Five cases (Fig. 4).

(3) Patients in whom there was no evidence as to the relationship between the liver and subphrenic abscesses. Two cases.

In none of these 17 cases was there any evidence to suggest that the primary illness gave rise to a subphrenic abscess which then spread into the liver. It appears that in the group (3) cases the liver and subphrenic abscesses developed independently of each other in two-thirds of the cases, and that in the remaining one-third a secondary liver abscess developed, and was responsible for a subphrenic abscess by direct extension of infection. In this second category the two abscesses were topographically related, whereas in the first category this was not necessarily so.

Barnard (1908) stated that a liver abscess usually spreads into the right extraperitoneal space, and that infection of the intraperitoneal subphrenic spaces from the liver is much less common. This is not supported by my findings, for only two out of the 21 patients studied had extraperitoneal abscesses.

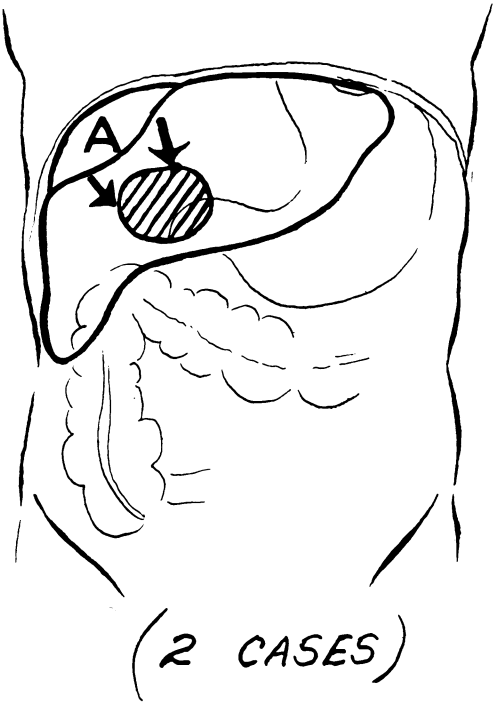

Fig. 2. Primary subphrenic abscess causing secondary liver abscess by direct spread of infection.

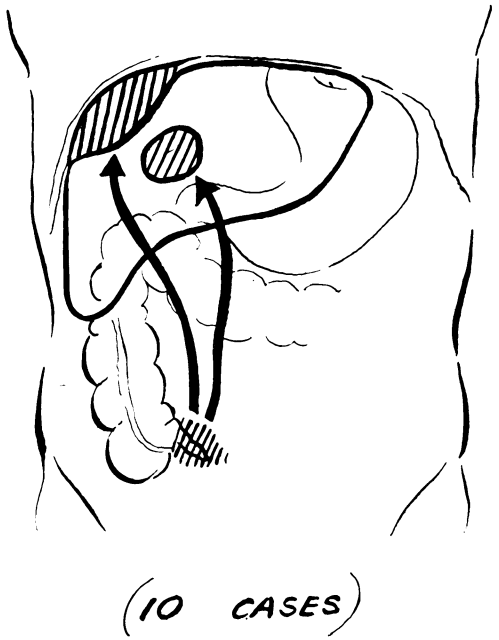

Fig. 3. Independent liver and subphrenic abscesses both secondary to acute appendicitis.

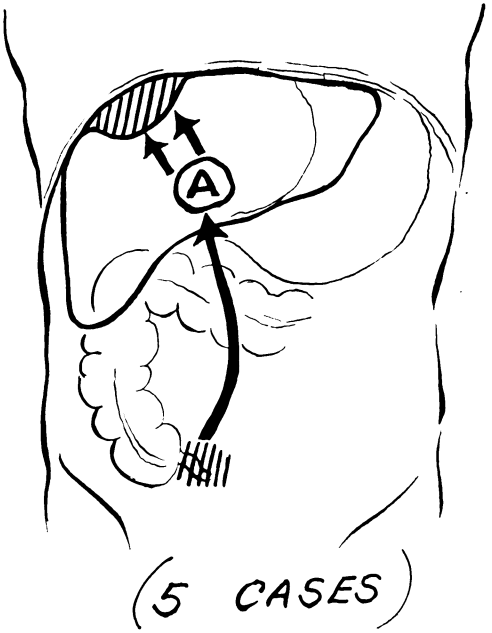

Fig. 4. Liver abscess due to acute appendicitis causing subphrenic abscess by direct spread of infection.

The liver was infected from the primary source of diseases in three different ways, by portal vein thrombosis and suppurative pylephlebitis (six cases), by suppurative cholangitis (two cases), or by direct extension of infection from a neighbouring focus, such as a perforated and adherent duodenal ulcer (two cases). There was no evidence as to how the liver was infected in seven cases, but it must have been in one or other of these three ways. Direct spread of infection to the liver usually produces a solitary abscess, whereas suppurative pylephlebitis and suppurative cholangitis usually result in multiple abscess formation.

In summary of the above it may be stated that infection is liable to spread from the liver to the subphrenic spaces, but not in the reverse direction. When a patient has both liver and subphrenic abscesses it is likely that the two abscesses have arisen as the result of disease elsewhere in the abdomen, but a primary liver abscess may have given rise to a secondary subphrenic one.

The method of spread of infection from the subphrenic region to the chest

There has been much discussion as to how infection spreads from the subphrenic spaces across the diaphragm to the chest, and two common misconceptions require correction. The first of these is that infection usually spreads across the diaphragm via lymphatic pathways, and the second is that serous pleural effusions are precursors of empyemata. The second of these will be considered first.

Serous effusions into the pleura frequently occur in patients with subphrenic abscesses, and should always make one suspect such an abscess in a patient with abdominal disease. Such an effusion was proved by aspiration, operation or necropsy examination in 48 (25.5 per cent.) out of the 188 patients, and was suggested in 13 others by radiological examination. These effusions are caused by inflammatory dilatation of the minute vessels under the diaphragmatic pleura. The diaphragm in these patients forms part of the wall of an abscess, and its blood vessels undergo the usual changes of inflammation. The serous pleural effusion which occurs is analagous to that which takes place into the knee joint when acute inflammation occurs near its capsule, or to the oedema which is found around an abscess in the soft tissues.

The pleural fluid is inflammatory, but not infective. It may be clear or cloudy. It was examined bacteriologically in 21 of the 48 cases, and in 18 of these no micro-organisms could be demonstrated either in films or on culture. In one case bacteria were seen on films, but the cultures were sterile. In only two cases could organisms be grown. The protein content, examined in five cases, varied between 1.2 per cent. and 3.3 per cent. The chief cellular constituents of 16 fluids examined were red blood cells, neutrophils and lymphocytes. A differential cell count was made on 13 of the fluids. In eight of these neutrophils predominated over

lymphocytes, in four lymphocytes were in excess of neutrophils, and in one the two types of cell were present in about equal proportions.

In no case studied did a serous effusion become purulent, unless a subphrenic abscess ruptured through the diaphragm into it, when the serous effusion was suddenty converted into an empyema. The only exception was a patient in whom open drainage of the serous effusion was performed. Table V indicates the incidence of serous pleural effusion in patients with and without suppuration in the chest. It is seen that the incidence was almost identical in the two groups, and that it was in fact slightly less in patients who developed an empyema than in those without an infection in the chest.

TABLE V

INCIDENCE OF SEROUS PLEURAL EFFUSION IN SUBPHRENIC ABSCESS PATIENTS WITH AND WITHOUT SUPPURATIVE COMPLICATIONS IN THE CHEST

Type of Case	Number	Number with serous Effusion	Percentage with serous Effusion
Cases without intrathoracic suppuration	134	35	26.1
Cases with any form of intrathoracic suppuration	54	13	24.1
Empyema or pyopneumothorax	39	9	23.1
Bronchial fistula	22	6	27.3
Suppurative pneumonitis	16	6	37.5
Perforation of diaphragm	22	7	31.8
TOTAL	188	48	25.5

From what has been said it is clear that serous pleural effusions are not precursors of intrathoracic suppuration and that the mechanism of their production is entirely different. The way in which infection spreads across the diaphragm still requires explanation.

Many believe that infection spreads across the diaphragm by lymphatic pathways. This route is suggested by histological studies of the lymphatic vessels of the diaphragm, which indicate that there is a special absorptive mechanism at this site, and by numerous experiments which prove that bacteria, red blood cells and particulate matter of many varieties are absorbed from the peritoneal cavity via these lymphatic vessels, and that they then pass on to the mediastinal nodes and thoracic duct with great rapidity.

The work of Vincentini (quoted by Sappey, 1874), Bizzozero and Salvioli (1876), Muscatello (1895), MacCallum (1903), Florey (1927), and Allen (1936) has shown that in the region of the diaphragm the peritoneal mesothelium is separated from the endothelium of the subperitoneal lymphatic vessels only by a thin fenestrated basement membrane, and that these lymphatic vessels communicate freely with the subpleural

diaphragmatic plexus by numerous connecting vessels passing through the muscular substance of the diaphragm. Bolton (1921) found that in most mammals the subpleural lymphatic plexus empties into two sets of efferent vessels, anterior (ventral) and posterior (dorsal). The anterior set drains into the internal mammary and anterior mediastinal nodes, and thence into the right lymphatic duct, and establishes communications with the thoracic duct. The posterior set drains into the cisterna chyli and thoracic duct, running en route through one or two upper lumbar and posterior mediastinal nodes. Bolton, and most subsequent observers, found the anterior route of drainage to be much the more important.

The astonishing speed of absorption of bacteria, red blood cells and particulate matter from the peritoneal cavity via the routes described above is indicated by the experiments of many workers, including von Recklinghausen (1863), Muscatello (1895), Durham (1897), MacCallum (1903), Buxton and Torrey (1906), Danielson (1907), Bolton (1921), Cunningham (1922 and 1926), Siperstein and Sansby (1923), Florey (1927), Levi (1927), Florey and Witts (1928), K. Paterson Brown (1928), Higgins and Graham (1929), Higgins, Beaver and Lemon (1930), Allen

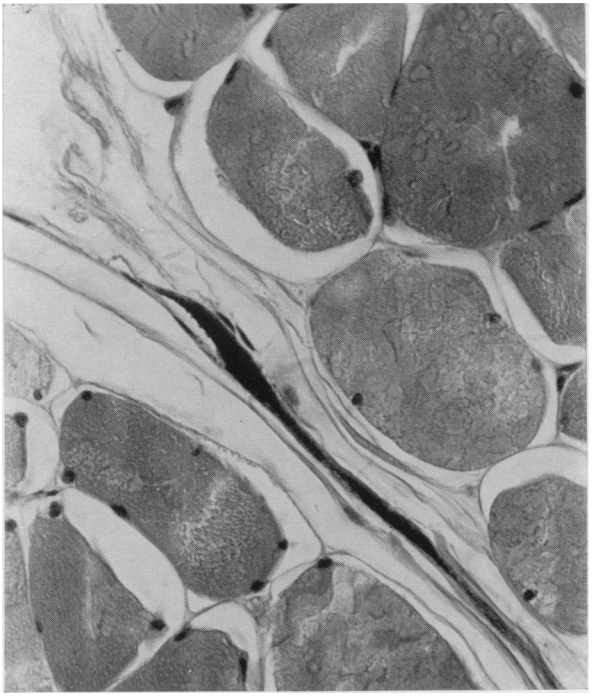

Fig. 5. Section of the diaphragm of a cat, showing a lymph channel filled with graphite absorbed from the peritoneal cavity.
Reproduced from original of Fig. 2, G. M. Higgins, M.G. Beaver and W. S. Lemon (1930), "*Amer. J. Anat.*" **45**, 137.

(1936), and Hahn, Miller, Robscheit-Robbins, Bale and Whipple (1944). A few only of these experiments need be mentioned here. MacCallum (1903) found the anterior mediastinal nodes to be stained bright pink two hours after injecting carmine into the peritoneal cavity of dogs. Cunningham (1922) injected a mixture of washed sheep's red blood cells, large unfiltered carmine particles, and small filtered lamp black granules into the peritoneal cavity of cats, and was able to demonstrate all three substances in the anterior mediastinal nodes within three minutes. Bolton (1921) demonstrated sarcinae and staphylococci, killed by heating on three successive days, in considerable numbers in the anterior mediastinal nodes of cats two hours after intraperitoneal injection. Colloidal silver stained these nodes brown in five minutes and black later. Danielson (1907) recovered colon bacilli from the lymph stream within 14 minutes. Higgings, Beaver and Lemon (1930) after intraperitoneal injection in the dog, demonstrated graphite in the subpleural lymphatic vessels in two to three minutes, in the mediastinal nodes in five minutes, and in the thoracic duct in 30 minutes. They made the important observation that when particulate matter was injected into the lower quadrant of the peritoneal cavity it was rapidly forced into the space between the liver and the diaphragm and appeared in the lymphatic channels of the latter in two to three minutes. Hahn *et al.* (1944) injected red cells labelled with radioactive iron into the peritoneal cavity of dogs and proved that the red cells were rapidly absorbed unchanged into the lymphatic vessels, chiefly of the diaphragm. They traced the labelled cells through the lymph nodes of the thorax to the thoracic duct, and thence to the large veins. The lymphatics of the anterior mediastinum stood out as beaded vessels filled with blood, and the lymph nodes of the hila of the lungs and of the upper mediastinum were swollen and cherry red, or mottled red and grey. Figs. 5-9, after Higgins, Beaver and Lemon (1930) and K. Paterson Brown (1928) are representative of this work.

Despite the above quoted work, a study of the 54 patients who developed suppuration in the chest gave no indication that the 72 suppurative complications which developed in these patients were caused by extension of infection along lymphatic pathways. No evidence of inflammation of the diaphragmatic, internal mammary or mediastinal nodes was reported, and no patient developed suppurative mediastinitis. This complication occurred in only two out of 1,380 cases (0.14 per cent.) collected by Ochsner and DeBakey (1938). These findings do not support the thesis that infection commonly spreads to the chest by lymphatic pathways.

The reason why chest infection developed was usually obvious, and was commonly due to one of three catastrophies, namely (1) rupture of the subphrenic abscess through the diaphragm ; (2) transpleural transdiaphragmatic drainage of the abscess ; and (3) penetrating thoracoabdominal wounds. The findings are shown in Table VI.

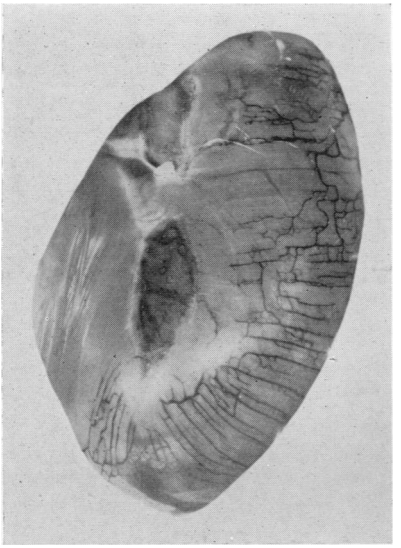

Fig. 6. Pleural surface of the right diaphragm of a dog, 45 minutes after peritoneal injection. The lymphatics are well filled.

Reproduced from original of Fig. 1, *G. M. Higgins, M. G. Beaver and W. S. Lemon* (1930), "*Amer. J. Anat.*" **45,** 137.

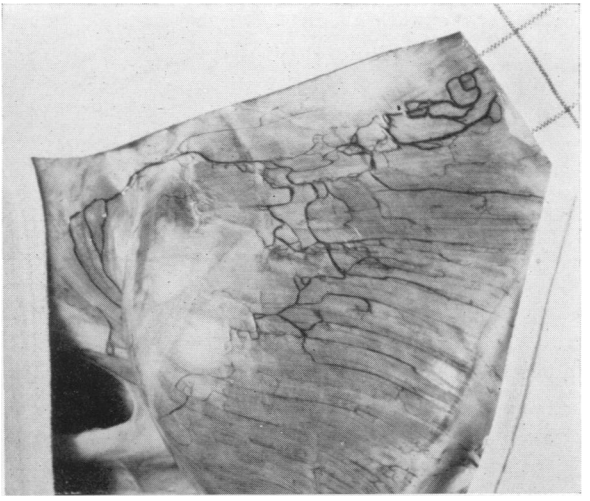

Fig. 7. Portion of the pleural surface of the diaphragm in a dog, following peritoneal injection. Channels coursing towards the oesophagus lead to a large lymph node near the spleen.

Reproduced from original of Fig. 3, *G. M. Higgins, M. G. Beaver and W. S. Lemon* (1930), "*Amer. J. Anat.*" **45,** 137.

TABLE VI

CAUSE OF INFECTION IN THE CHEST OCCURRING IN CONSEQUENCE OF SUBPHRENIC ABSCESSES IN 54 PATIENTS WITH 72 INTRATHORACIC SUPPURATIVE COMPLICATIONS

Cause of intrathoracic complication	Number of Patients	Per cent.
*Perforation of diaphragm	18	33.3
Perforation of diaphragm suspected, but not proved	2	3.8
Transpleural drainage of subphrenic abscess	12	22.2
Penetrating thoraco abdominal wounds	6	11.1
Diagnostic aspiration	1	1.9
Open drainage of serous pleural effusion	1	1.9
Pulmonary actino mycosis	1	1.9
Inhalation lung abscess	2	3.8
No cause found	11	20.4
TOTAL	54	100.3

* The patient with pulmonary actino mycosis also had a perforated diaphragm, but the pulmonary condition was considered to be the cause of the empyema. Another empyema was due to transpleural drainage, but there was also a perforated diaphragm. These two cases in which there was a perforated diaphragm are shown under other headings. Two other patients in whom the diaphragm was perforated did not have suppuration in the chest. Thus although perforation of the diaphragm occurred in 22 cases only 18 of them are shown in this table and Table VIII.

The table indicates that a cause for infection in the chest was not found in only 11 patients. Four of these had bronchial fistulae and it is almost certain that rupture through the diaphragm had occurred, but it was not demonstrated. It is probable, therefore, that all except seven patients had a gross cause for chest infection.

The incidence of the 72 suppurative complications which occurred in the chest in these 54 patients is shown in Table VII, while the causes of these complications are detailed in Table VIII.

TABLE VII

INCIDENCE OF INTRATHORACIC SUPPURATIVE COMPLICATIONS OCCURRING IN 54 OUT OF 188 PATIENTS AS A DIRECT CONSEQUENCE OF SUBPHRENIC ABSCESS

Complication	Number of Patients	Per cent.
Empyema	35	18.6
Pyopneumothorax	4	2.1
Suppurative pneumonitis without bronchial fistula	7	3.7
Bronchial fistula	22	11.7
Suppurative pericarditis	4	2.1
Mediastinal abscess	0	—
Perforation of diaphragm	22	11.7
Total number of patients who developed one or more of these complications	54	28.7
Total number of patients studied	188	

TABLE VIII

CAUSES OF 72 INTRATHORACIC SUPPURATIVE COMPLICATIONS OCCURRING IN 54 PATIENTS SUFFERING FROM SUBPHRENIC ABSCESS

Intrathoracic complication	Cause											
			Surgical causes									
	No cause demonstrated	Perforation of diaphragm	Transpleural drainage	Diagnostic aspiration	Open drainage of serous pleural effusion	Penetrating Wounds	Lung suppuration	Empyema	Lung suppuration and empyema	Inhalation	Actinomycosis	TOTAL
Empyema and pyopneumothorax	6	10	12	1	1	6	2	–	–	–	1	39
Suppurative pneumonitis with bronchial fistula	*4	5	–	–	–	–	–	13	–	–	–	22
Suppurative pneumonitis without bronchial fistula	1	3	–	–	–	–	–	–	–	2	1	7
Suppurative pericarditis	–	–	–	–	–	–	2	1	1	–	–	4
TOTAL	11	†18	12	1	1	6	4	14	1	2	2	72

* These four bronchial fistulae were almost certainly caused by perforation of the diaphragm by the subphrenic abscess, but proof is lacking.

† See footnote to Table VI which explains how figure of 18 was obtained.

A study of the 11 patients shown in Table VIII in whom no cause for the chest infection was proven indicates that in four of them, all with bronchial fistulae, the fistula almost certainly followed rupture of the abscess through the diaphragm, for there was no empyema, and a bronchial fistula could not have developed in any other way. The patient with suppurative pneumonitis, but without a bronchial fistula, probably developed his infection as the result of inhalation or pulmonary collapse. Lymphatic infection is most unlikely to have caused these five complications. In the six patients of this group who developed an empyema lymphatic infection is obviously a possible cause, but perforation of the diaphragm may well have occurred, and have been responsible in five of them. Two of these six patients died, but an autopsy was performed on only one of them. This patient had a growth surrounding the cardiac end of the stomach, a right suprahepatic subphrenic abscess, collapse and consolidation of the right lung, and an empyema on the right side. There was no perforation of the diaphragm, and no operation for drainage had been performed. The pleura might have been infected from the lung, or as the result of lymphatic spread from the subphrenic abscess. The other five patients may well have had perforations of the diaphragm, for such could easily have been missed at operations for drainage.

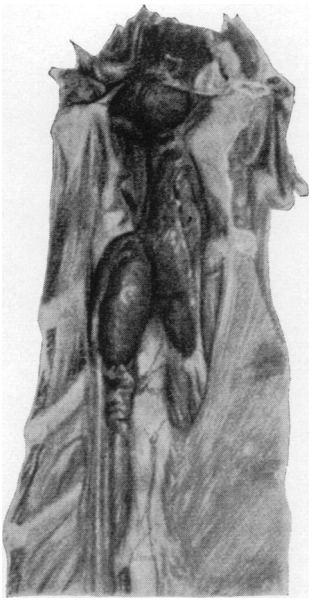

Fig. 8. Glands on deep aspect of sternum of dog stained by colloidal silver after intraperitoneal injection.
Reproduced from original of Fig. 386, *K. Paterson Brown* (1928), "*Brit. J. Surg.*" **15,** 538.

I wish now to discuss briefly the principal causes that were found to account for infection in the chest. As already stated the three most frequent of these were rupture of the subphrenic abscess through the diaphragm, transpleural drainage of the abscess, and penetrating thoraco-abdominal wounds.

Rupture of subphrenic abscesses through the diaphragm

This event is fairly common, and is the most important cause of infection spreading to the chest.

Direct evidence of perforation of the diaphragm was obtained at operation on necropsy in 22 out of the 188 patients, an incidence of 11.7 per cent., and this was considered to be the cause of intrathoracic suppuration in 18 out of 54 patients (33.3 per cent.). As stated above rupture of a subphrenic abscess through the diaphragm almost certainly accounted for four cases of bronchial fistula in addition, so that perforation of the diaphragm probably occurred in 26 patients. Ochsner and DeBakey (1938) give the incidence as 27.7 per cent. for 1,380 collected cases of subphrenic abscess.

When a subphrenic abscess ruptures through the diaphragm an empyema is produced if the diaphragmatic pleura remains free, but a bronchial fistula occurs if the pleura has already become adherent. Occasionally a localised diaphragmatic empyema is first formed and subsequently

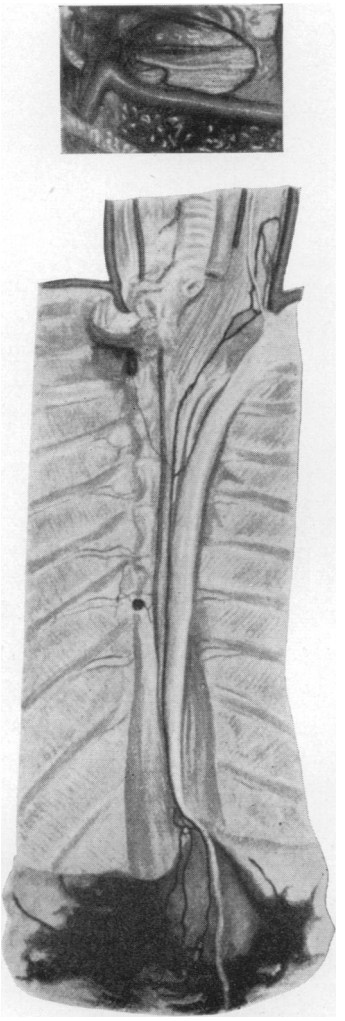

Fig. 9. Absorption of colloidal silver from peritoneal cavity of rabbit, showing subpleural plexus and whole length of thoracic duct.
Reproduced from original of Fig. 383, *K. Paterson Brown* (1928), "*Brit. J. Surg.*" **15,** 538.

bursts into the lung. An empyema resulting from rupture of a subphrenic abscess into a free pleura may subsequently perforate the lung to cause a bronchial fistula, so that all fistulae are not due to rupture of the subphrenic abscess directly into the lung. Rupture of a gas-containing subphrenic abscess into a free pleura may cause a pyopneumothorax, and this was the sequence of events in one of the four pyopneumothoraces in this series. Two were caused by transpleural drainage of subphrenic abscesses, and one by a sucking thoracoabdominal wound.

The perforation of the diaphragm may be very large. Beye (1932) said its size might vary from that of a probe to an aperture several centimetres in diameter, and that in most cases it would readily allow of the insertion of an index finger. In one case studied by me the perforation measured 6 cms. in diameter, in a second 2 cms. in diameter, in a third 1 cm. in diameter, and in a fourth the opening admitted one finger. The perforation is produced as the result of inflammatory necrosis of a considerable area of the diaphragm. Fig. 10 illustrates an infected hydatid cyst of the liver which has ruptured through the diaphragm into the lower lobe of the right lung. The perforation in the diaphragm is a large one.

Transpleural drainage of subphrenic abscesses

Transpleural drainage accounted for 12 cases of infection in the chest, and was its second commonest cause. In 10 of these patients an empyema followed the operation, while the other two developed a pyopneumothorax. This is a very serious indictment against this method of draining

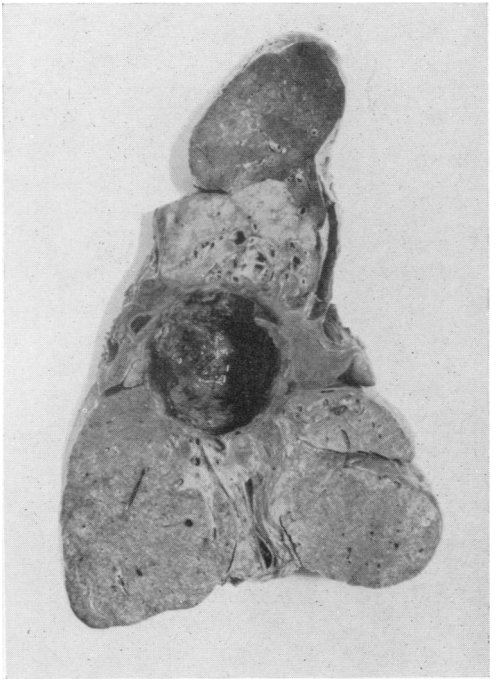

Fig. 10. Infected hydatid cyst of right lobe of liver which has ruptured through the diaphragm into the lower lobe of the lung. The diaphragm is destroyed over a considerable area. The lower lobe of the lung is completely consolidated and the site of multiple abscesses.

Reproduced from specimen A 99.7 *in the pathological section of the Hunterian Museum by kind permission of Dr. L. W. Proger. The specimen was presented to the Royal College of Surgeons of England in* 1949 *by the Royal Australasian College of Surgeons.*

subphrenic abscesses. I wish to stress the fact that pleural adhesions, whether caused by the abscess, or produced artificially by the surgeon, afford no certain protection against infection of the pleural cavity, even if the line of incision through the pleura is confined to the adherent area. Barnard (1908) recorded five cases in which pleural adhesions gave way when the diaphragm descended after the abscess was drained. This happened twice at the time of operation, and three times a day or two later. Grueneisen (1903) found that in 28 per cent. of subphrenic abscesses opened through a normal pleura, and in 35 per cent. of those in which there existed sterile fibrinous adhesions, empyema developed despite the various procedures employed to protect the pleura. Doherty and Rowlands (1931) also stress the failure of pleural adhesions to prevent the occurrence of empyema after transpleural drainage of subphrenic abscesses. The mortality of trans-serous methods of drainage is far higher than that of the extra-serous method, and the latter should be employed whenever possible.

Diagnostic aspiration of subphrenic abscesses

This procedure carries a small but definite risk of conveying infection to the pleura. Diagnostic aspiration was performed on 82 out of the 188 patients studied. In 60 patients it was carried out in the ward and in 30 in the operating theatre (in 12 patients it was performed in both places). One patient in whom aspiration was performed in the ward developed an empyema which was considered to have been caused by the procedure, but there was no evidence that aspiration performed in the operating theatre was ever responsible for such an infection. Table IX shows my findings for diagnostic aspiration performed in the ward, and also the incidence of empyema given by three other observers who did not state the circumstances of aspiration.

TABLE IX
INCIDENCE OF EMPYEMA FOLLOWING DIAGNOSTIC ASPIRATION OF SUBPHRENIC ABSCESS

Authority	Number of patients aspirated	Number who developed empyema
Barnard (1908)	18	1
Overholt and Donchess (1935)	21	1
Neuhoff and Schlossmann (1942)	17	0
Present series (performed in ward)	60	1
TOTAL	116	3

In Barnard's patient no pus was obtained at aspiration, but three and a half hours later the patient died suddenly, and at necropsy it was found that one and a half pints of turbid offensive pus had passed through the puncture track in the diaphragm from the right subphrenic space into the pleura. The procedure may, therefore, be dangerous even when no pus is withdrawn. Apart from the danger of infecting the pleura

diagnostic aspiration performed in the ward is very unreliable, for pus was found in only 14 out of the 60 patients (23 per cent.) in the present series, and in my view it is a method of investigation which should not be employed.

Penetrating thoracoabdominal wounds

Wounds of this nature were the cause of 10 out of the 188 subphrenic abscesses studied, and were responsible for intrathoracic infection in six out of 54 patients (11.1 per cent.). Five of the six patients developed an empyema and one a pyopneumothorax, as shown in Table VI. The high incidence of penetrating wounds is due to the fact that many of the cases studied occurred during the 1939-45 War.

Summary

The evidence indicates that spread of infection from distant parts of the abdomen to the subphrenic region is usually due to the movement of infected peritoneal fluid within the peritoneal cavity, but that less commonly it is due to spread via the portal vein, with a resultant pylephlebitis. It is probable that two forces account for this movement of peritoneal fluid to the subphrenic region, gravity when the patient is recumbent, and hydrostatic pressure when he is upright. Once infected fluid reaches any part of the diaphragm it is likely to be spread over its surface by the constant movement occurring between the diaphragm and its related viscera. No evidence was found to suggest that spread of infection within the abdomen takes place via lymphatic pathways.

Infection appears to spread readily from the liver to the subphrenic spaces, especially the intraperitoneal ones, but not in the reverse direction.

The spread of infection across the diaphragm to the chest is almost always caused by a sudden catastrophe, such as rupture of the subphrenic abscess through the diaphragm, transpleural drainage of the abscess, or a penetrating thoracoabdominal wound. No evidence was found to inculpate lymphatic spread in the conduction of infection to the chest.

Serous pleural effusions result from the out-pouring of an inflammatory, but non-infective, exudate caused by dilatation of the minute vessels under the diaphragmatic pleura. They have a different mode of origin from infective complications in the chest, and they should not be considered as harbingers of empyemata.

ACKNOWLEDGMENTS

My thanks are due to the members of the staffs of the undermentioned hospitals for granting me access to the case records of patients admitted under their care. These hospitals are : St. Bartholomew's, Charing Cross, Guy's, The London, The Middlesex, St. George's, St. Thomas's, University College, The Westminster, The Brompton, The London Chest, Harefield County, Horton Emergency, Addenbrooke's and St. Stephen's. My thanks are also due to Mr. F. J. Sambrook Gowar for allowing me

to have the records of three of his patients. I am indebted to Blackwell Scientific Publications Ltd. for allowing me to quote from the text of a monograph entitled "Subphrenic Abscess" which is in process of publication. To Prof. Lambert C. Rogers I owe especial thanks for his encouragement and stimulation in the preparation of this lecture. I would like to thank also Miss June Williams, Mr. R. K. Marshall and Mr. L. Williams for their help and co-operation in the preparation of slides, and Miss June Williams for the drawings of Figs. 1, 2, 3 and 4. My thanks are also due to Dr. L. W. Proger for allowing me to illustrate Specimen A 99.7 in the pathological section of the Hunterian museum.

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HEROES OF THE EXAMINATION BOOK

3. SIR CHARLES NAPIER (1786-1860)

IT IS NOT unlikely that the two visits to the College of Captain Charles Napier caused some surprise even there, for Black Charley's appearance was so singular that it commonly excited wonder " amounting almost to consternation. One of his acquaintances described him as being " about fourteen stone, stout and broad built ; stoops from a wound in his neck, walks lame from another in his leg, turns out one of his feet ; and has a most slouching, slovenly gait, a large round face with black bushy eyebrows, a double chin, scraggy, grey, uncurled whiskers and thin hair ; wears a superfluity of shirt collar and small neck-handkerchief ; always bedaubed with snuff, which he takes in immense quantities ; usually his trousers far too short and wears the ugliest pair of old shoes he can find."

Napier was born on 6th March, 1786 and entered the navy thirteen years later. His first appearance before the Court of Examiners on 1st September, 1809, was the result of an encounter with the French sloop *Diligente* on 5th September of the previous year when although his thigh was broken, he refused to leave the deck until the fall of the mainmast of his ship ended the engagement. As a result of the report made by the Court he was placed on half pay but a life of semi-retirement did not suit him and in 1810 " by way of amusement " he went to Spain where, during, the Battle of Busaco on 27th September, he was again wounded. At the Court of Examiners held on 1st March, 1811, his injury was deemed not equal to the loss of a limb and he at once returned to active service.

Thenceforth his was a varied career. During a brief residence in Paris in 1818 he helped to finance the launching of iron steamers on the Seine, a venture that, on its failure in 1827, left him a comparatively poor man. In 1833 he accepted the command of the Portuguese fleet and for his services was made Viscount Cape St. Vincent. On his entry into Lisbon he was hailed as the liberator of Portugal and Don Pedro himself conferred on him the Grand Cross of the Order of the Tower and the Sword. He was knighted in 1840 and acclaimed as a national hero until an unsuccessful expedition to the Baltic in 1853 somewhat marred his reputation.

On being elected Member of Parliament for Southwark he retired from naval life and, having become afflicted with the mania of letter-writing, amused himself until his death on 6th November, 1860, in writing bitter tirades against the service that he had adorned so spectacularly for almost sixty years.

The country had cause to be indebted to this gallant officer not only for his own daring feats but that, during his participation in the Peninsular War, he saved the life of his cousin, Charles James Napier, whose prospects of lasting renown were considerably enhanced on 18th May, 1844, by Mr. Punch's report on Foreign Affairs as follows :

> " It is a common idea that the most laconic military despatch ever issued was that sent by Caesar to the Horse Guards at Rome, containing the three memorable words *Veni, vidi, vice,* and perhaps until our own day no like instance of brevity has been found. The despatch of Sir Charles Napier, after the capture of Scinde, to Lord Ellenborough, both for brevity and truth, is, however, far beyond it. The despatch consisted of one emphatic word *Peccavi.*"