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Hepatic Hydrothorax

Studies to Determine the Source of the Fluid and Report of Thirteen Cases

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THE ASSOCIATION of pleural effusion with ascites has been mentioned commonly in the literature, usually in association with solid ovarian tumors, the Tait-Demons-Meigs' syndrome (1–3) and with hepatic cirrhosis (4–10). Meigs' work, calling attention to the triad of benign ovarian tumor, ascites, and hydrothorax, is well-known. He demonstrated that both peritoneal and pleural effusions could be cured by simply removing the ovarian tumor (1). Meigs, Armstrong, and Hamilton (11) showed that particulate matter was transported from the peritoneal to the pleural space, but not in the opposite direction; they also thought that hydrothorax was probably caused by transfer of ascitic fluid to the pleural space via diaphragmatic lymphatics.

The association of hydrothorax with hepatic cirrhosis has also been recognized for many years. Vedel and Puech (4) reported that hydrothorax was present in 8 to 9% of their cirrhotic patients, occurring more commonly on the right side. Christian (5) reported a case of cirrhosis with a large bloody left pleural effusion and ascites. At autopsy, no intrinsic pleural disease was found. Frothingham (6) described a case of cirrhosis, complicated by massive right hydrothorax. The case was of interest for, when frequent thoracenteses were done, ascitic fluid did not accumulate. However, when pleural fluid decreased after a pleural space infection, frequent abdominal paracenteses became necessary. In total, over 467 liters of chest fluid and 214 liters of ascitic fluid were removed. Tinney and Olsen (7), in a review of 274 cases of pleural effusion, attributed 8 or 3.4% to cirrhosis. In an autopsy study of 600 cases of cirrhosis Mc-

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Kay, Sparling, and Robbins (8) found hydrothorax in six patients, thought to be caused by cirrhosis, an incidence of 1%. Ascites was present in all of the six patients. Tumen (9) states the incidence of hydrothorax in cirrhosis is 10%. Morrow, Kantor, and Armen (10) reported a single case, which was the only one found in 250 cases of cirrhosis, giving an incidence of 0.4%.

From these observations there is little doubt that hydrothorax can occur with cirrhosis in the absence of manifest chest disease. In most of the reported cases, ascites was present. It has been postulated by many that in this condition, as in Meigs' syndrome, the transport of ascitic fluid into the pleural space accounts for at least some of the hydrothorax.

In this paper we will report 13 additional examples of hepatic hydrothorax and, from studies on one case, will attempt to show that transport of ascitic fluid to the pleural space via the diaphragmatic lymphatics accounts for at least some of the hydrothorax.

Materials and Methods

Chart Study

After Case 1 had been studied, the charts of 200 consecutive patients with a final diagnosis of hepatic cirrhosis, hospitalized between January 1, 1957 and June 30, 1962, were reviewed in a search for cases of pleural effusion. Patients were considered to have hepatic hydrothorax when other causes of pleural effusion, such as cardiac failure, neoplasm, pleural and pulmonary infections, trauma, and pulmonary infarction, could be reasonably excluded.

Special Studies on Case 1

COMPARISON OF FLUIDS

Samples of peritoneal and pleural fluid were obtained within minutes of each other, then analyzed for various components by standard laboratory techniques.

INDIA INK STUDIES

Ten milliliters of sterile India ink were injected into the peritoneal space. Twenty-four hours later, samples of peritoneal and pleural fluid were obtained. Ten-milliliter samples of fluid were centrifuged; smears were prepared and stained by the Papanicolaou technique. This test was repeated approximately 1 month later with the ink being placed in the pleural space.

PNEUMOPERITONEUM

After preliminary upright films of the chest and abdomen were obtained, 1,000 ml of carbon dioxide were introduced into the peritoneal space through a polyethylene catheter. The roentgenograms were then repeated.

INJECTION OF RADIOIODINATED ALBUMIN INTRAVENOUSLY, INTRAPERITONEALLY, AND INTRAPLEURALLY

Polyethylene catheters were placed into the right pleural and peritoneal spaces and into a peripheral vein, or in the case of the last experiment, into the brachial artery. After samples of fluid and blood had been obtained to determine the background counts, approximately 60 μc of radioiodinated serum albumin (Albumotope-I^{131}, Squibb Laboratories) were injected, the first experiment intravenously, the second experiment intraperitoneally and the third experiment intrapleurally. Serial samples of fluids were then obtained for 50 hr. Two-milliliter samples of whole blood, peritoneal and pleural fluids were counted in a well scintillation counter for 10 min, and the count corrected for decay to the day of injection. Albumin concentrations were measured in each fluid and blood hematocrit reading determined. The counts per gram of albumin in each (plasma, pleural and peritoneal fluids) were calculated thus:

Plasma:

Counts/gram albumin

$$= \frac{\text{Counts/tube}}{2\ (\text{1-hematocrit})(\text{albumin in gram/milliliter plasma})}$$

Pleural and Peritoneal Fluids:

Counts/gram albumin

$$= \frac{\text{Counts/tube}}{2\ (\text{albumin in gram/milliliter of fluid})}$$

The counts per gram of albumin from each were plotted against time.

TABLE 1. Summary of Thirteen Cases of Hepatic Hydrothorax

No.	Age	Sex	Race	Serum Albumin	Location of Hydrothorax	Character of Fluid		Type of Cirrhosis	Remarks
						Pleural	Peritoneal		
				g/100 ml					
1	66	F	W	2.5	R	Clear yellow; AFB culture negative; no malignant cells; TP, 0.9 g/100 ml	Clear yellow; no malignant cells; TP, 0.7 g/100 ml	Portal	Esophageal varices present; died 21 months after discovery of the effusion; autopsy: diaphragm intact; lung, pleural, ovaries, normal
2	58	F	W	1.5	R	Not removed	No malignant cells; TP, 0.7 g/100 ml	Liver biopsy showed only fatty metamorphosis	Spider angiomata and hepatomegaly; 3 years later, effusion gone
3	46	M	W	2.1	R	AFB culture negative; no malignant cells; TP, 0.6 g/100 ml	Not removed	Probably portal	Alcoholic patient died after 2 weeks of hepatic failure
4	50	M	W	2.5	R	AFB culture negative; no other studies	Not removed	Probably portal	Alcoholic patient with icterus and edema; died after 2 weeks
5	45	F	W	3.7	R	Not removed	AFB culture negative; no other data	Portal	Alcoholic patient with cirrhosis proved at biopsy; no follow-up
6	71	M	W	3.3	R	Clear yellow; TP, 0.6 g/100 ml	Straw colored; no malignant cells; TP, 0.9 g/100 ml	Probably portal	Alcoholic patient with icterus and edema; venous pressure, 130 mm Hg; saline, circulation time (arm to tongue) 12 sec; died after 2 weeks from hepatic failure
7	49	F	W	2.0	Bil	Clear yellow fluid from both sides; AFB culture negative; no malignant cells	Not removed	Probably portal	Alcoholic patient with esophageal varices; died after 2 months from hemorrhage and hepatic failure
8	57	F	W	1.6	L	Not removed	Not removed	Probably poral	Alcoholic patient who died on second hospital day

TABLE 1. *(Continued)*

No.	Age	Sex	Race	Serum Albumin	Location of Hydrothorax	Character of Fluid		Type of Cirrhosis	Remarks
						Pleural	Peritoneal		
				g/100 ml					
9	63	F	W	2.5	R	Clear yellow; AFB culture negative; no malignant cells	Not removed	Portal	Alcoholic patient with cirrhosis proved at biopsy; died in 2 weeks from hemorrhage and hepatic failure
10	40	F	N	2.5	R	Not removed	Not removed	Probably portal	Alcoholic patient discharged in 3 weeks; no follow-up study
11	75	M	W	3.4	L	Serosanguinous; AFB culture negative; no malignant cells; WBC, 82/mm^3; RBC, 280/mm^3; TP, 2.9 g/100 ml	AFB culture negative; no malignant cells; TP, 2.8 g/100 ml	Portal	Alcoholic patient with cirrhosis proved at biopsy; died 11 months later following portocaval shunt; pleural effusion still present
12	64	M	N	2.3	R	Not removed	Yellow; AFB culture negative; TP, 1.7 g/100 ml	Probably portal	Alcoholic patient with ascites and edema; discharged after 2 weeks; no follow-up study
13	29	F	W	3.0	Bil	Not removed	Not removed	Portal	Alcoholic patient with cirrhosis proved at biopsy; discharged after 4 weeks no follow-up study

Results

Case Studies

Table 1 summarizes the 13 cases which we accepted as examples of hepatic hydrothorax. Table 2 indicates the locations and incidence of the pleural effusions in the 200 cases of cirrhosis reviewed.

Table 2. Location and Incidence of Hydrothorax in 200 Cases of Cirrhosis

	Number	Incidence	Number with Ascites
		%	
Right pleural effusion	8 (66.7%)	4	8 (100%)
Left pleural effusion	2 (16.7%)	1	2 (100%)
Bilateral effusions	2 (16.7%)	1	2 (100%)
	—	–	—
Total	12 (100%)	6	12 (100%)

Special Studies on Case 1

COMPARISON OF FLUIDS

Table 3 compares the peritoneal and pleural fluids obtained simultaneously. The

Table 3. Comparison of Pleural and Peritoneal Fluids from Case 1

	Pleural Fluid	Peritoneal Fluid
Character	Yellow, clear	Yellow, clear
Glucose, mg/100 ml	118	109
Urea, mg/100 ml	38	39
Chloride, mEq/liter	100	100
Bicarbonate, mEq/liter	25	22
Sodium, mEq/liter	123	124
Potassium, mEq/liter	5.0	4.7
Total proteins, g/100 ml	0.90	0.70
	1.2	0.90
Albumin, g/100 ml	0.50	0.34
	1.30	0.90
	0.90	0.70
Globulins, g/100 ml	0.40	0.36
	0.30	0.20
Total cholesterol, mg/100 ml	42	11
	21	16
Total lipids, mg/100 ml	455	175
	105	35
	280	210

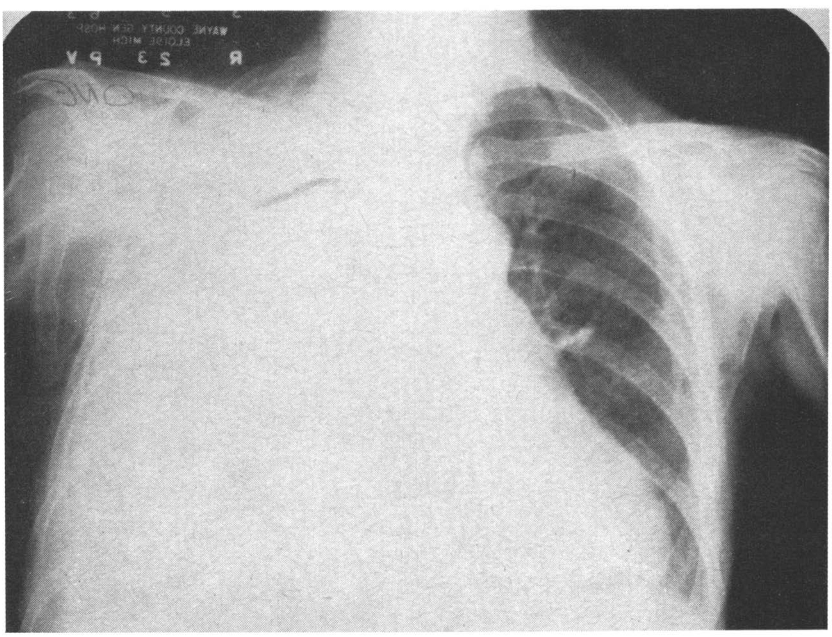

Figure 1. *Case 1.* Huge right pleural effusion with leftward shift of the heart and mediastinum.

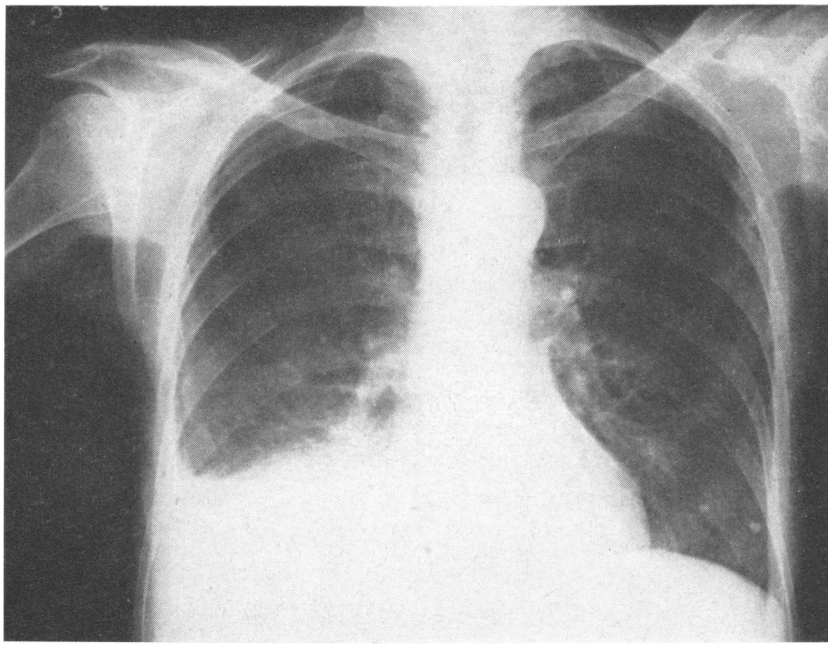

FIGURE 2. *Case 2.* Small right pleural effusion and calcified granulomata on the left.

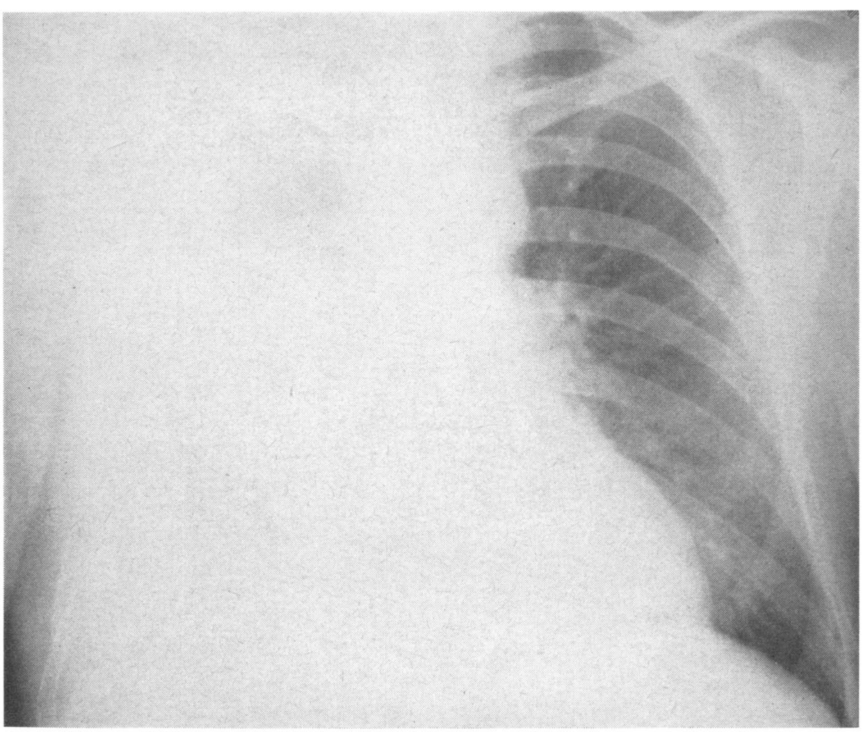

FIGURE 3. *Case 3.* Huge right pleural effusion with slight leftward shift of the heart and mediastinum.

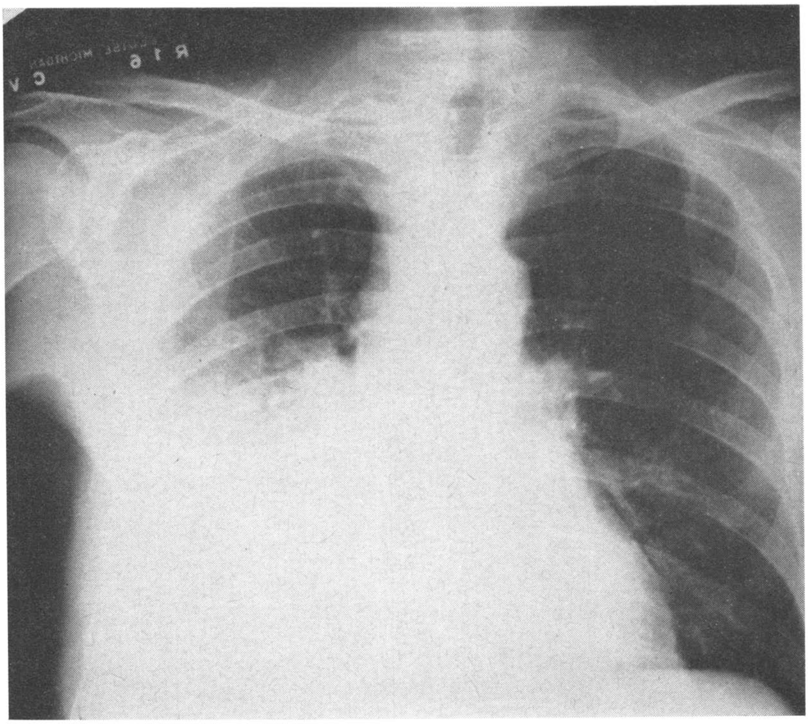

FIGURE 4. *Case 4.* Large right pleural effusion.

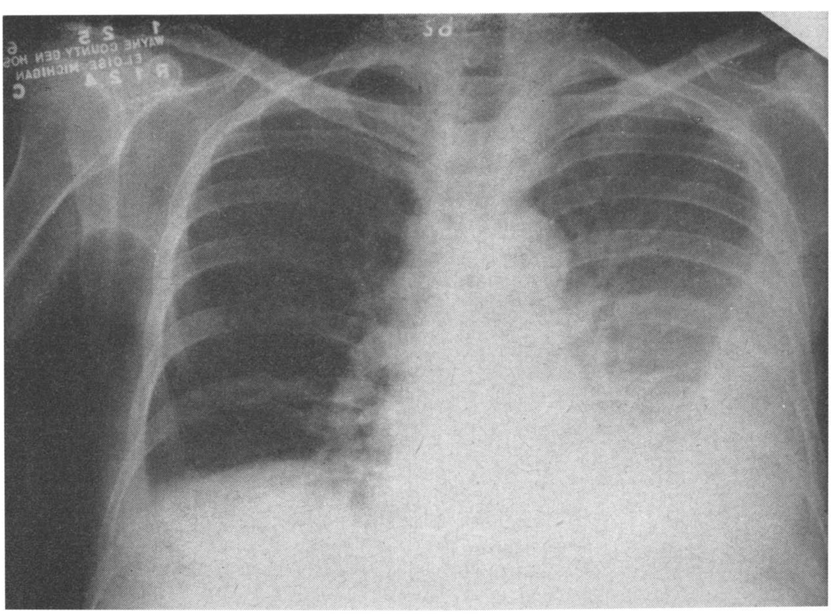

FIGURE 5. *Case 7.* Bilateral pleural effusion.

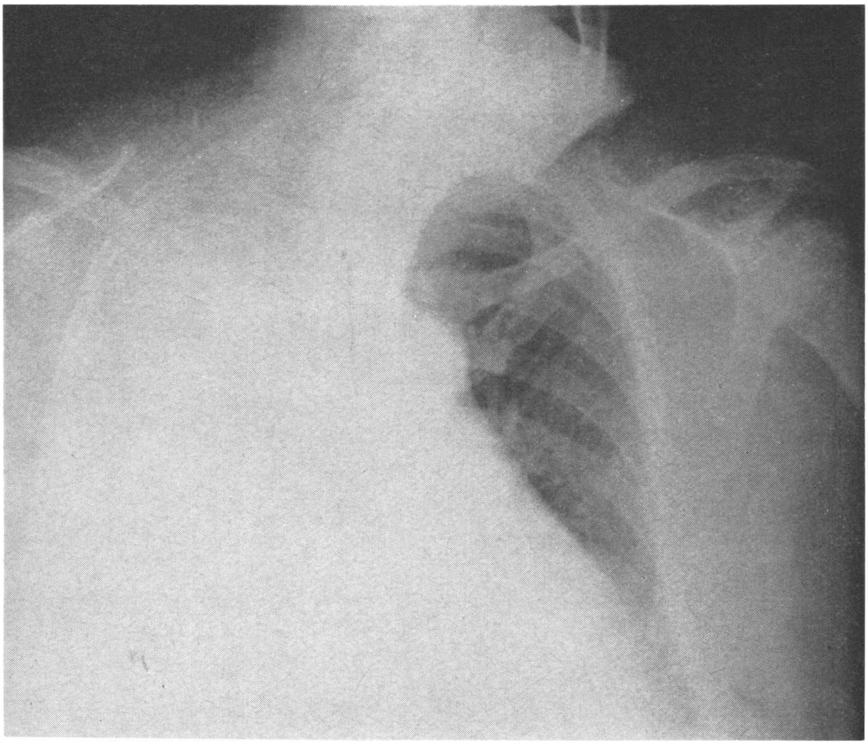

FIGURE 6. *Case 9.* Huge right pleural effusion.

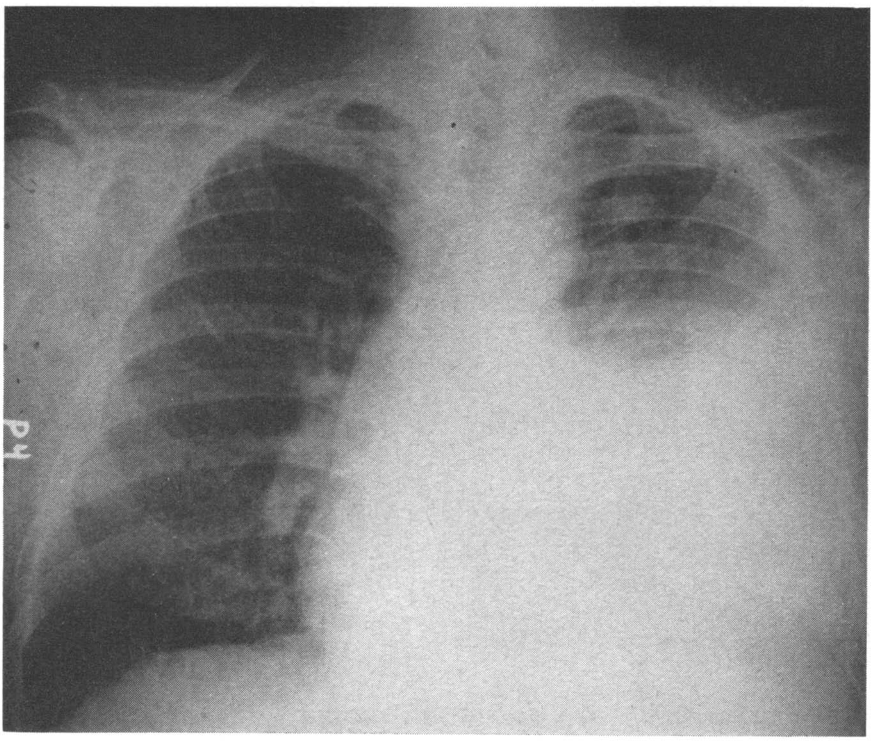

FIGURE 7. *Case 11.* Large left pleural effusion with rightward shift of the heart and mediastinum.

fluids are similar in most respects. Both meet the criteria for transudates (total protein less than 3 g/100 ml). The total protein is slightly higher in the pleural fluid due to a larger albumin fraction.

INDIA INK STUDIES

After the intraperitoneal injection of India ink, smears prepared from pleural fluid showed the macrophages contained many carbon particles (Figure 8). Carbon particles could not be seen in smears of peripheral blood obtained at the same time. After intrapleural injection of the ink, there was no qualitative increase in carbon particles in the peritoneal fluid macrophages, although some carbon particles were still present, presumably from the previous intraperitoneal injection. Samples of peritoneal fluid obtained before and after the intrapleural ink injection had the same optical densities, supporting the impression that no transfer of carbon took place.

PNEUMOPERITONEUM

The roentgenograms taken after intraperitoneal injection of carbon dioxide showed no transfer of gas from the peritoneal to pleural space.

INTRAVENOUS INJECTION OF RADIOIODINATED ALBUMIN

Figure 9 illustrates that after the intravenous injection of radioiodinated serum albumin, radioactivity appeared more rapidly and in greater concentration in peritoneal fluid than in pleural fluid. At 26 hr the plasma radioactivity was only 1.6 times that of peritoneal fluid, but was still 4.7 times that of pleural fluid. By 51 hr the radioactivity per gram of albumin in plasma and peritoneal fluid was equal, but radioactivity per gram of albumin of plasma was still 2.2 times that of pleural fluid.

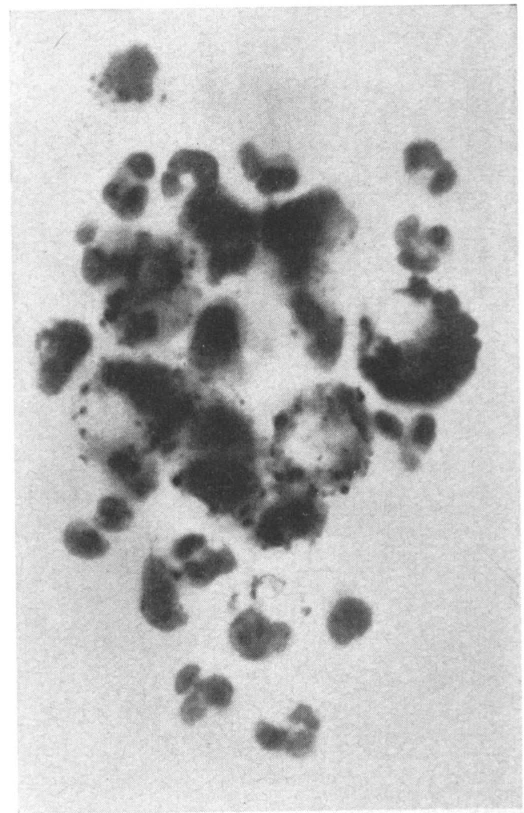

FIGURE 8. Photomicrograph of pleural fluid from *Case 1* showing carbon particles in the macrophages. Papanicolaou stain, × 1,400.

INTRAPERITONEAL INJECTION OF RADIOIODINATED ALBUMIN

Figure 10 demonstrates that after intraperitoneal injection of radioiodinated serum albumin, radioactivity appeared in the pleural fluid and plasma at about the same time and in the same concentration. At 25 hr the radioactivity per gram of albumin in plasma and pleural fluid was nearly identical, in contrast to results of intravenous study, where at 25 hours the plasma radioactivity per gram of albumin was nearly five times that of pleural fluid. These results suggest that radioactive iodine-tagged albumin was entering the right pleural space directly from the peritoneal space. After intraperitoneal injection equilibrium between the three spaces oc-

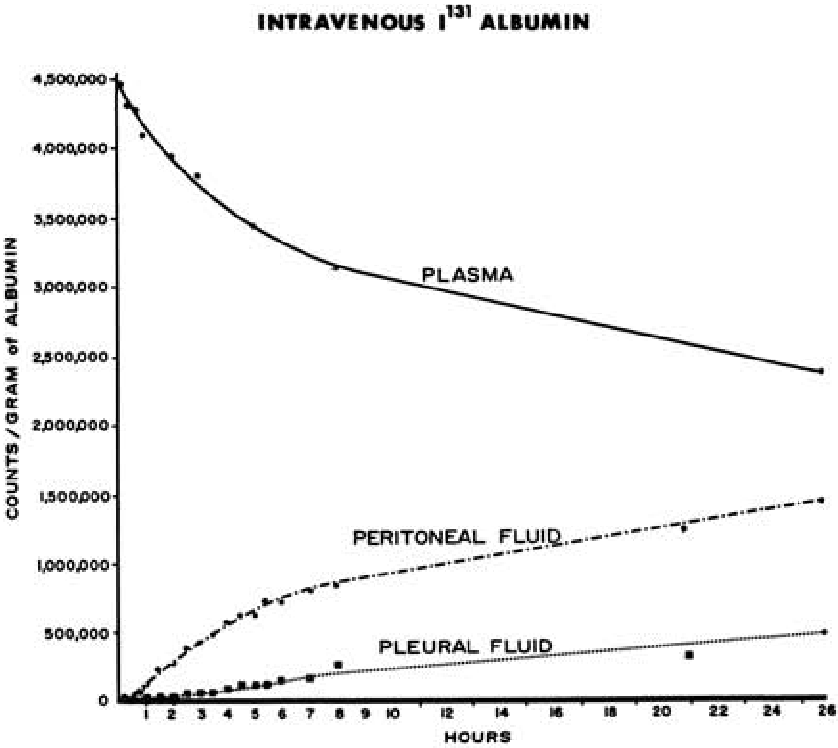

FIGURE 9. *Case 1.* Radioactivity of body fluids after the intravenous injection of radioiodinated serum albumin.

curred more slowly, for at 50 hr the peritoneal fluid contained 4.4 times as much radioactivity per gram of albumin as pleural fluid and 3.6 times as much radioactivity per gram of albumin as plasma.

INTRAPLEURAL INJECTION OF RADIOIODINATED ALBUMIN

Figure 11 indicates that after injection of radioiodinated serum albumin into the right pleural space, radioactivity appeared more rapidly and in higher concentration in the plasma than in the peritoneal fluid. At 25 hr radioactivity per gram of albumin in plasma was 4.9 times that in the peritoneal fluid. This suggests that radioactivity was transported to the peritoneal space via plasma, and there was no direct passage of tagged albumin from the pleural to the peritoneal space. Equilibrium of the three spaces was slower after intrapleural than either intravenous or intraperitoneal injection. At 50 hr the radioactivity per gram of albumin in pleural fluid was still 16.8 times that of plasma and 56 times that of peritoneal fluid.

DISCUSSION

The source of chest fluid found in association with cirrhosis is unknown. Morrow, Kantor, and Armen (10) suggested that the fluid may come from the blood stream as a result of hypoalbuminemia or hypertension in the azygous and hemiazygous systems or be secondary to ascites either by direct passage of the fluid through a diaphragmatic defect or by transport of ascitic fluid into the pleural space through the diaphragmatic lymphatics. Hypoalbuminemia as a sole cause can easily be eliminated, for there are many cirrhotic patients and persons with malabsorption syndromes who

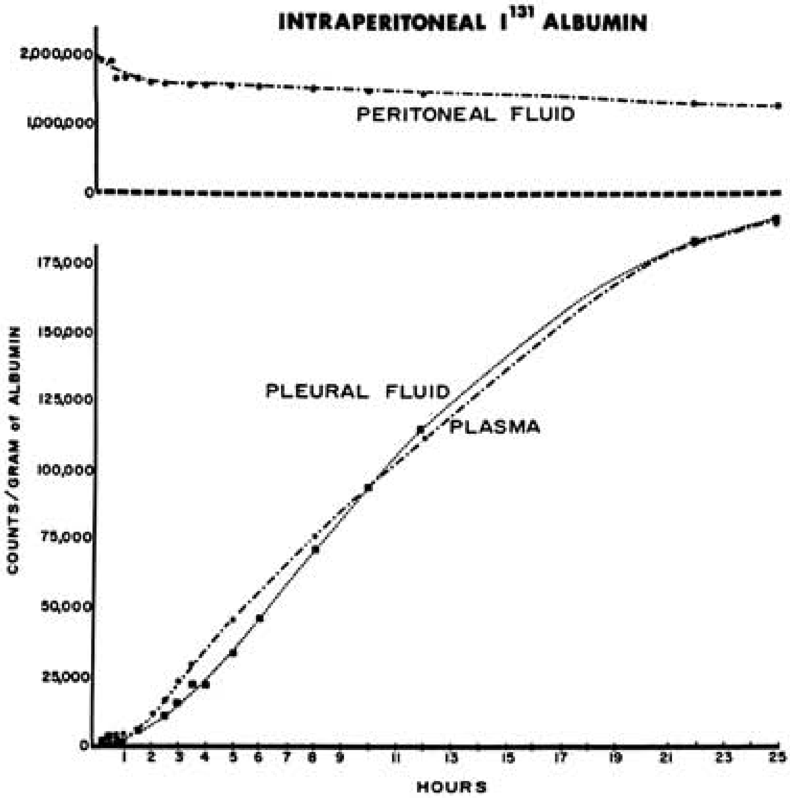

FIGURE 10. *Case 1.* Radioactivity of body fluids after the intraperitoneal injection of radioiodinated serum albumin.

have low serum albumin levels but never develop hydrothorax. Of the 200 cases of cirrhosis reviewed from this hospital, serum albumin levels were obtained in 179. Figure 12 indicates there was a significant difference between the mean serum albumin levels for those cirrhotic patients without ascites and the two other groups composed of patients with ascites alone and with both ascites and hydrothorax. However, there was no significant difference between the groups with just ascites and the group with ascites and hydrothorax.

In addition to hypoalbuminemia, if azygous hypertension is present due to collaterals between this system and the portal system, transudation of fluid into the chest, or more likely decreased absorption of pleural fluid, might occur. However, this explanation cannot be used to explain hydrothorax occurring in Meigs' syndrome where there is no portal hypertension. Furthermore, with this mechanism, you would expect bilateral effusion, which occurred in only 17% of our cases. Although it seems clear hypoalbuminemia and azygous hypertension play some role in the etiology of hepatic hydrothorax, these factors alone cannot adequately explain all cases.

The more probable explanation is that hydrothorax is derived directly from the peritoneal fluid. Ascites is present in Meigs' syndrome and in nearly all cases of cirrhosis when hydrothorax is recognized. All of our 13 cases had ascites, and though there have been reports of hepatic hydrothorax occurring in the absence of ascites (12), these are rare. We have assumed, therefore, that

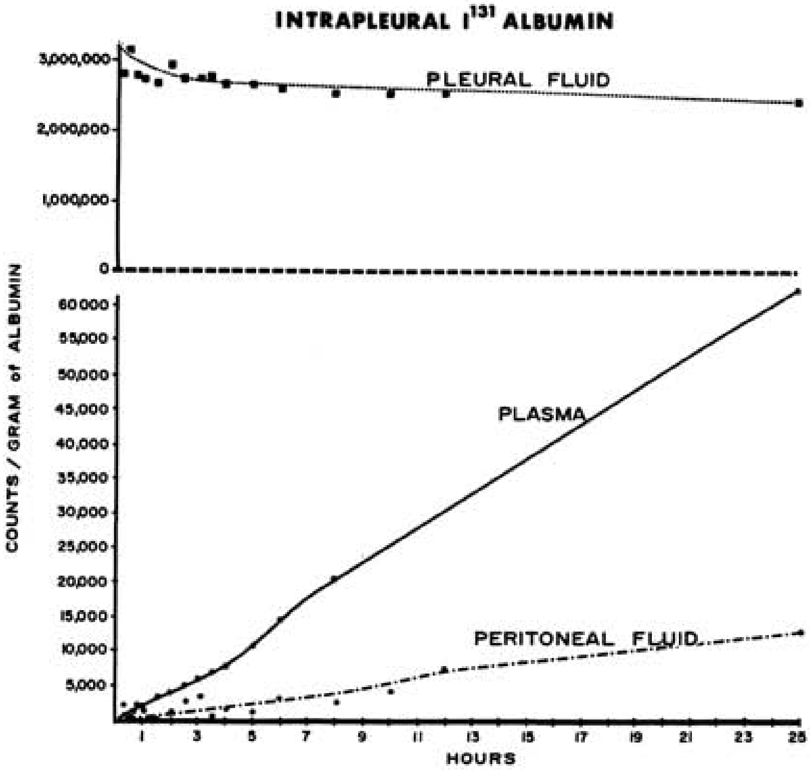

FIGURE 11. *Case 1.* Radioactivity of body fluids after the intrapleural injection of radioiodinated serum albumin.

ascitic fluid passes into the pleural space either through a defect in the diaphragm or via the lymphatic channels that penetrate it.

In our Case 1, we strongly considered the possibility of there being a defect in the right diaphragm providing a direct communication between the peritoneal and pleural spaces. However, our studies tended to eliminate this possibility, for if there was direct mixing of fluids through such a defect the following would be expected:

1. The fluids should be identical. Though our analysis showed the pleural and peritoneal fluids were similar, total proteins, albumin, cholesterol, and total lipids were consistently higher in the pleural fluid (Table 3).

2. An indicator substance introduced into one space should soon be present in equal concentrations in the other space. In our studies this did not occur with either India ink or radioiodinated serum albumin. Furthermore, it was demonstrated that the flow was unidirectional from peritoneal to pleural space.

3. Gas introduced into the peritoneal space should pass freely into the pleural space. This did not occur in our patient when carbon dioxide was given intraperitoneally.

These interpretations were confirmed by autopsy, which showed no gross defect in the right diaphragm. The remaining possibility is that the peritoneal fluid is transported to the pleural space through the diaphragmatic lymphatics. This is supported by the demonstration of unidirectional transport of carbon particles from peritoneal to pleural spaces and by the

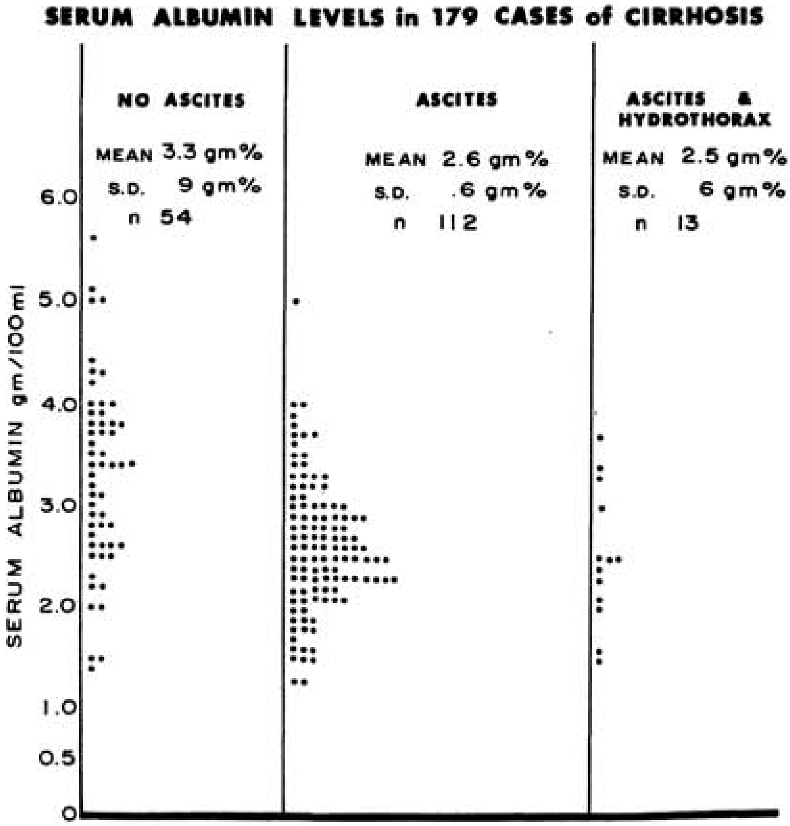

FIGURE 12. The differences between the means of the group with no ascites and the other two groups are significant ($P < 0.001$). There is no significant difference between the means of the two groups with ascites.

isotope studies which suggest that tagged albumin is transported directly from the peritoneal to the pleural space and not in the opposite direction.

Evidence has been presented in the literature suggesting that particulate matter and proteins are removed from the peritoneal cavity almost exclusively by the subdiaphragmatic lymphatics. After the fluid has passed into the lymphatic plexus on the peritoneal surface of the diaphragm, it is carried through the diaphragm into a similar lymphatic plexus on the pleural surface. The lymph then passes from the diaphragmatic nodes into collecting ducts, which run with the internal mammary vessels on both sides of the sternum until they reach the anterior mediastinal nodes. From these nodes the efferent vessels pass most commonly toward the right side, where eventually they enter the right subclavian or jugular veins or occasionally unite to form a right lymphatic duct which then enters the venous system (13). Lemon and Higgins (14) demonstrated that, in the dog, graphite injected into the peritoneal space appeared on the pleural surface of the diaphragm within 3 to 4 min. Although other alternate pathways exist, they estimated that 80% of the injected material was carried to the blood stream through the diaphragmatic and retrosternal lymphatics. They noted that the lymphatics were more abundant in the right than in the left diaphragm, an observation which has also been made in man (15). Courtice and associates (16–18) showed that protein labeled with T-1824 (Evan's blue) was absorbed from the peritoneal and pleural spaces almost exclusively by the same lymphatic pathways. In addition they demonstrated that when retrosternal lymphatics were ligated in rabbits, guinea pigs and rats, pleural effusion occurred.

In humans limited data has been presented indicating that particulate matter and colloids pass from the peritoneal to pleural spaces. The works of Meigs, Armstrong, and Hamilton (11) with carbon particles has been cited, and it has been demonstrated that radioactive colloidal gold passes from peritoneal to pleural spaces. However, this data also showed prompt passage from pleural to peritoneal spaces, suggesting the possibility of a diaphragmatic defect (19). Unfortunately, data on plasma radioactivity was not included in the report. In several cases of hepatic hydrothorax, Patek (20) reports that T-1824 or India ink placed into the peritoneal cavity was detected in the pleural fluid. Flow in the reverse direction was not seen.

Our data, suggesting that hydrothorax was not formed exclusively from plasma or as the result of a diaphragmatic defect, is compatible with the theory that chest fluid is the result of transdiaphragmatic transport of ascitic fluid by the lymphatics. The location where the fluid leaves the lymphatic system and enters the pleural space is not known, neither has the mechanism causing such exudation been clearly demonstrated. In addition, the site of absorption of fluid from the pleural space in man is not know. Courtice and Simmonds (17) have demonstrated that in the pleural and peritoneal spaces of dogs, protein is absorbed almost exclusively by the lymphatics. In the dog it has been shown that most of the protein-containing fluid in the pleural space is absorbed through the lower mediastinal pleural folds and the parietal intercostal pleura. Eventually, the fluid absorbed from the pleural space reaches the venous system through the thoracic or right lymphatic duct (21). Although Mayerson and his associates (22) have shown that in the dog substances with a molecular weight greater than 2,300 do not pass through the walls of large normal lymphatics, they later showed that injury produced by various means would increase permeability of these large vessels. They further postulated that large molecules, such as proteins, can pass freely in both directions through the walls of the lymphatic capillaries and that compression of these channels in any manner would squeeze lymph out of the vessels.

The concept that hepatic hydrothorax is produced from peritoneal fluid transported to the pleural space by the lymphatics receives some limited support from the recent work of Dumont and Mulholland (23). They have demonstrated that when cirrhosis and ascites are present, lymph flow from the cannulated thoracic duct is increased with a range of 2.0 to 12 ml/min (normal less than 1.0 ml/min) and that the end pressure of the thoracic duct is elevated with a range of 15 to 70 cm of saline (normal 6 to 15 cm of saline). They also made the interesting observations that liver size in four patients and ascites in seven patients markedly decreased while the thoracic duct was vented. Dumont believes the increased thoracic duct flow is due to liver lymph and not to absorption of ascitic fluid, because the thoracic duct fluid showed higher concentrations of protein and red blood cells that the ascitic fluid. This may be correct, for previous work has indicated that the thoracic duct plays little part in drainage of the peritoneal cavity as most of the absorbed fluid enters the venous system through the right thoracic duct (13). If, however, we accept the concept that protein is absorbed from the peritoneal space almost exclusively by the subdiaphragmatic lymphatics, it is probable that flow through the absorbing lymphatics will increase when ascites is present. It is difficult to exclude the possibility that some of this fluid enters the thoracic duct. The difference in composition between the thoracic duct lymph and peritoneal fluid is to be expected, for the absorbed ascitic fluid would merely serve to dilute the lymph and not entirely to replace it.

Using the same line of reasoning, if the ascitic fluid is indeed transported to the pleural space through the lymphatics, the composition of the ascitic and pleural fluids might differ. Most studies have demonstrated that protein content of lymph from the thoracic duct is over 3 g/100 ml, with the albumin fraction usually being over 2 g/100 ml (24). Although data is limited one would also expect that lipid content would be higher in lymph than in transudates in serous cavities. Therefore, the protein and lipid content of the fluid originating in the peritoneal space, which has passed through the lymphatics and then entered the pleural space, might increase depending upon the permeability of the lymphatic vessels for protein and lipids. Though our analysis of the peritoneal and pleural fluids showed them to be similar, the total protein, albumin, total lipids and cholesterol fractions of the pleural fluid on all occasions were higher than the same fractions in the peritoneal fluid (Table 3).

Dumont, Clauss, Reed, and Tice (25) have extended their studies to patients with congestive heart failure. Here, again, they demonstrated that in five patients the thoracic duct was increased in diameter and lymph flow was increased 4 to 12 times normal. After venting the duct venous pressure fell and peripheral edema, ascites, dyspnea and orthopnea decreased. The mechanism responsible for these changes is not clear, however the reminder is timely that in congestive heart failure there is increased flow of lymph through the thoracic duct. It is possible the pleural effusions seen in congestive heart failure originate from the thoracic lymph and that some of the fluid may originally have been absorbed from the peritoneal space by the lymphatic pathways described. The greater number of lymphatics in the right diaphragm could explain the predominate right-sided location of cardiac hydrothorax. Origin of these effusions from lymphatics could also explain their high protein concentration.

If it is correct that hepatic hydrothorax is secondary to transdiaphragmatic transport of ascitic fluid in the lymphatics, the question must be answered why doesn't hydrothorax occur in all patients with ascites? Pleural effusion will not be manifest until the rate of formation of pleural fluid exceeds the rate of fluid absorption from the pleural space. In the pleural space it has been demonstrated that protein is absorbed by the lymphatics (17) and asssumed that water and solutes are exchanged freely with the plasma as they are in the peritoneal space (26). Even in normal man it is likely that pleural fluid is being constantly formed, but an equal rate of absorption prevents accumulation of fluid. Yamada (27) in a group of several hundred normal Japanese soldiers was able to aspirate a small amount of pleural fluid in 29% of the group when they were at rest. After heavy exercise he obtained pleural fluid from 70%, suggesting that temporarily the rate of formation had exceeded the rate of absorption. In the presence of ascites, we believe that rate of pleural fluid formation will be greater. If in addition hypoalbuminemia is present, the rate of absorption of water will be decreased. If the rate of formation exceeds the rate of absorption, hydrothorax will occur. The probability of hydrothorax occurring will be increased if there are local factors, such as azygous hypertension which impedes the absorption of water or obstruction or increased pressure in the thoracic lymphatics which reduces the absorption of protein. Dumont and Mulholland (23) feel there may be significant obstruction of the thoracic duct in the neck and that improvement noted after cannulation was due to relief of the obstruction. Lemming (28) suggested the occurrence of hydrothorax in Meigs' syndrome may be the "result of ineffective intrathoracic lymph drainage (the flow of lymph being more or less blocked due to earlier inflammatory processes within the thorax, for example, hilar tuberculosis)".

Stewart (29), assuming that protein in man is removed from the pleural space solely by the lymphatics, has shown with the use of T-1824 that on the average, 0.364 ml of fluid/kg of body weight is absorbed per hour by the lymphatics of one pleural cavity. Using a modification of the Stewart method and making an additional assumption that the volume of the pleural effusion remained constant, we have calculated from the experiment on Case 1 where tagged albumin was placed into the right pleural space that only 0.229 ml of pleural fluid/kg of body weight was absorbed by the lymphatics per hour (body weight 40 kg, 9.2 ml of pleural fluid absorbed per hour), suggesting that in this patient absorption of protein containing fluid from the pleural space was reduced. Although the mechanisms involved in the formation of hepatic hydrothorax still are not clear, it does seem reasonable to suggest that local factors affecting the rate of absorption of pleural fluid can determine whether or not the patient with ascites develops hydrothorax.

Summary

In 200 consecutive cases of cirrhosis, the incidence of hydrothorax has been shown to be 6%. The effusion occurred on the right side in 67%, bilaterally in 17%, and on the left side in 17%. All cases with hydrothorax had ascites. In one case studied in detail, it was concluded that radioiodinated serum albumin and carbon particles passed from the peritoneal to the pleural spaces, most probably via the diaphragmatic lymphatics. Transportation of ascitic fluid to the pleural space via this route appears to be the best explanaton for the occurrence of hydrothorax with hepatic cirrhosis and with ovarian tumors which are accompanied by ascites. The occurrence of hydrothorax in some but not all cases of ascites can best be explained by systemic and local factors controlling the absorption of fluid from the pleural space.

Summario in Interlingua

In 200 consecutive casos de cirrhosis, le incidentia de hydrothorace esseva 6 pro cento. Le effusion occurreva al latere dextere in 67 pro cento del casos, bilateralmente in 17 pro cento, e al latere sinistre in 17 pro cento. In omne le casos con hydrothorace, ascites esseva presente. In un caso, que esseva studiate in detalio, il esseva concludite que radio-iodate albumina seral e particulas de carbon passava ab le spatios peritonee ad in le spatios pleural, le plus probabilemente via le lymphaticos diaphragmatic. Transporto de liquido ascitic verso le spatio pleural per le mentionate via pare explicar le melio le occurrentia de hydrothorace con cirrhosis hepatic e con tumores ovarian que es accompaniate de ascites. Le occurrentia de hydrothorace in certe sed non omne casos de ascites se explica le melio per fatores local e general que regula le absorption de liquido ab le spatio pleural.

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