THE PREPONDERANCE OF RIGHT HYDROTHORAX IN CONGESTIVE HEART FAILURE*

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ALTHOUGH there has been a strong clinical impression that right hydrothorax is more common than left hydrothorax when gross congestive heart failure occurs, recently this point of view has been questioned. In a survey by Bedford and Lovibond,[1] these authors were led to the conclusion that although right hydrothorax was more common in cases showing mitral stenosis, combined right and left heart failure and auricular fibrillation, the occurrence of left hydrothorax was favored by hypertension, left heart failure and normal rhythm. Owing to the strong impression we have had that right hydrothorax was much more common in congestive heart failure, regardless of the underlying cause, the following study was made to determine the actual facts. Consecutive cases of congestive heart failure showing hydrothorax were selected, after eliminating those presenting extraneous factors, such as pleurisy with effusion, active rheumatic fever, significant nephritis, blood dyscrasias, neoplasm, hepatic disease, etc. The purpose of this was to include for consideration only those suffering from clear-cut cardiovascular disease with congestive failure.

The first method of approach was to review 75 clinical cases of congestive heart failure that required thoracentesis. In each case the side of the chest tapped was the only one in which fluid appeared to be present or was the one that seemed to show the larger degree of hydrothorax, where some disproportion in the two sides existed. This decision was made by the ordinary methods of bedside examination with or without the aid of roentgen-ray. The second analysis consisted of comparing the roentgen-ray findings in 52 consecutive cases of congestive heart failure with hydrothorax. In no instance had thoracentesis been performed in this group, and the relative degree of hydrothorax on the two sides was estimated by the roentgenologist. The basis of the third study consisted in determining the amount of fluid in the two pleural cavities in 110 cases of congestive heart failure that came to postmortem examination. The above three analyses ought to serve as controls for each other and should give convincing evidence if they all agree in their conclusions.

Of the 75 cases of hydrothorax requiring thoracentesis (table 1), 35 had rheumatic heart disease, 22 had a significant degree of coronary sclerosis and 18 had hypertensive heart disease. Owing to the tendency for hydrothorax to recur following thoracentesis, no more than two chest taps from

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TABLE I

Hydrothorax in Congestive Heart Failure, Thoracentesis

Heart disease		Rheumatic	Coronary	Hypertensive	Total
Site of hydrothorax	R*	28	16	11	55
	L	3	1	4	8
	B	4	5	3	12
Total cases		35	22	18	75
Site with auricular fibrillation	R	20	4	1	25
	L	1	0	1	2
	B	(R>L) 1	2	0	3
		(L>R) 2	0	0	2
Chest taps	R	45	22	14	81
	L	4	2	4	10
	B	(R>L) 2	5	3	10
		(L>R) 2	0	0	2
Total taps		57	34	24	115
Average amt. fluid removed	R	1000 c.c. (45)	1050 c.c. (22)	1050 c.c. (14)	1050 c.c.
	L	1100 c.c. (4)	1050 c.c. (2)	1000 c.c. (4)	1050 c.c.
	BR	1100 c.c. (4)	1300 c.c. (5)	1200 c.c. (3)	1200 c.c.
	BL	1150 c.c. (4)	550 c.c. (5)	800 c.c. (3)	850 c.c.

* R=right, L=left, B=bilateral, BR=bilateral R>L, BL=bilateral L>R.

any one case were included in this study, regardless of whether both were performed on the same side or one on each of the two sides. Auricular fibrillation was present in 32 of this series of cases, 24 being in the rheumatic group, six in the coronary and only two in the hypertensive group. Twenty-five of these 32 cases were aspirated from the right chest only, two from the left chest alone and in five bilateral taps were done with a greater amount of fluid removed from the right cavity in three of these. In the rheumatic group 57 thoracenteses were performed on the 35 cases, 49 times on the right side and eight times on the left. In 45 instances the right chest alone was aspirated and in only four the left alone. There were four instances in which both sides were tapped. It is of interest that the amount of fluid removed at each thoracentesis was almost the same no matter which side was tapped. When a similar review was made of the 22 cases of coronary heart disease it was found that 34 chest taps were performed, 27 on the right side and seven on the left, 22 on the right side alone and two on the left alone. In five instances bilateral paracenteses were done although in each instance the right hydrothorax was greater than the left. It seems significant that

while the average amount of fluid aspirated, where only one side was explored, was 1050 c.c., in the five cases in which both sides were explored an average amount of only 550 c.c. was obtained from the left side and 1300 c.c. from the right. In the last group, consisting of 18 cases of hypertensive heart disease, 24 chest taps were performed, 17 on the right and seven on the left, 14 on the right side alone and four on the left alone. Three bilateral aspirations were done, and in each instance the amount of fluid removed from the right pleural sac was greater than that removed from the left, the average being 1200 c.c. from the right and 800 c.c. from the left. In summary, among the 115 thoracenteses performed on 75 cases of congestive heart failure, 93 were done on the right chest and 22 on the left, 81 on the right chest alone and 10 on the left alone. In the 12 instances where both sides were explored, only twice was the amount of fluid removed from the right side found to be less than that removed from the left side.

The second approach toward determining the distribution of hydrothorax was made on 52 consecutive cases which had had no previous thoracenteses and were analyzed from the roentgenological evidence alone (table 2). Of this group the 18 with rheumatic heart disease presented pure right hydrothorax in nine instances, pure left hydrothorax in three, and in six fluid occurred bilaterally and about equally on the two sides. In the 12 cases of coronary artery disease, right unilateral hydrothorax was present in three, and in the other nine fluid was present on both sides, predominantly right in four, predominantly left in two, and about equally in three. The 16 cases of hypertensive heart disease showed fluid in the right thorax only in six, and all the remaining presented fluid bilaterally, more on the right in four, more on the left in two, and about equally in four. No definite tendency

TABLE II

Hydrothorax in Congestive Heart Failure, Radiological Examination

Heart disease		Rheumatic	Coronary	Hypertensive	Misc.	Total
Site of hydrothorax	R	9	3	6	2	20
	L	3	0	0	1	4
	B	(R>L) 0	4	4	1	9
		(R=L) 6	3	4	1	14
		(L>R) 0	2	2	1	5
Total		18	12	16	6	52
Site with Aur. Fib.	R	5	1	2	0	8
	L	1	0	0	0	1
	B	(R>L) 0	1	0	1	2
		(R=L) 4	1	1	0	6
		(L>R) 0	0	0	1	1

to a preponderance of fluid in either side of the thorax occurred in six cases of miscellaneous types of heart disease. Thus, of this entire series, 20 cases evidenced pure right hydrothorax, four pure left, with the remaining 28 presenting fluid on the two sides about equally, i.e. predominantly right in nine, predominantly left in five, and with no discernible difference in 14. Auricular fibrillation was present in 18 instances and of these 10 showed more fluid in the right chest, two in the left and in six it appeared to be equal. Unilateral hydrothorax in this group occurred approximately five times more often on the right side than on the left, and when the hydrothorax was bilateral there appeared no definite tendency for either side to predominate.

In the third study, 110 cases of congestive heart failure presenting hydrothorax at postmortem examination were analyzed (table 3), and the deductions were based on the amounts of fluid determined by direct measurement. It was realized that the agonal state might influence the formation of a terminal hydrothorax of limited degree, when the circumstances were favorable to such, and transudates of less than 300 c.c. were arbitrarily excluded from the study. A considerable number of these cases had had thoracenteses performed prior to death, and a quantitative analysis of the fluid so removed has been compiled for consideration.

The rheumatic group presented unilateral right hydrothorax twice, unilateral left three times, and in 50 the fluid was distributed bilaterally with the greater amount on the right in 44, on the left in four, and in two equally. In two of the three instances in which fluid occurred on the left side alone there was complete obliteration of the right pleural cavity by fibrous adhesions. Signs of left heart failure had dominated the clinical picture in five of the 55 cases, and in one of these fluid was found only on the left side, while on the other four it occurred bilaterally and predominantly right (3) or equal (1). Pulmonary infarction was present in 25 of this group and complicated left hydrothorax in three instances and right hydrothorax in one. In 21 cases so complicated, fluid was found in both cavities with the greater amount in the right in 20 of these. Prior to death auricular fibrillation had existed in 38 of the rheumatic patients, and of this number one showed fluid on the right side alone at autopsy, two on the left side alone and 35 presented fluid bilaterally with that on the right predominating in 33. The average amount of fluid found in the right pleural sac, in this rheumatic group, was 550 c.c. and that in the left sac was 350 c.c. Of similar interest is the fact that in 27 of this group of 55 cases, in which 73 clinical thoracenteses had been performed (57 right, 16 left), an average amount of 900 c.c. of fluid had been removed from the right thorax and 700 c.c. from the left thorax.

In this study, 25 cases of coronary heart disease presented hydrothorax entirely on the right in two instances, entirely on the left in one and in 22 it was bilateral with the right side predominating in 18, the left in three, and neither in one. Fibrous adhesions had completely obliterated the right pleural cavity in the one instance in which the fluid was limited to the left side. Nine of the 25 cases had presented the usual signs of marked left heart

TABLE III

Hydrothorax in Congestive Heart Failure, Post Mortem

Heart disease		Rheumatic	Coronary	Hypert.	Luetic	Total
Number		55	25	23	7	110
Average age		46	66	61	53	54
Site of hydrothorax	R	2	2	1	1	6
	L	3	1	1	0	5
	B (R>L)	44	18	18	5	85
	B (R=L)	2	1	0	0	3
	B (L>R)	4	3	3	1	11
Site with lt. ht. failure	R	0	0	0	1	1
	L	1	1	1	0	3
	B (R>L)	3	5	9	3	20
	B (R=L)	1	1	0	0	2
	B (L>R)	0	2	2	1	5
Site with pul. infarction	R	1	0	0	0	1
	L	3	0	0	0	3
	B (R>L)	20	6	3	2	31
	B (R=L)	1	0	0	0	1
Site with aur. fib.	R	1	0	0	0	1
	L	2	0	0	0	2
	B (R>L)	33	4	3	0	40
	B (L>R)	2	0	1	0	3
Average of fluid at PM	R	550 c.c.	550 c.c.	Above three groups considered together.		
	L	350 c.c.	400 c.c.			
Fluid removed during life	Cases	27	26			
	Taps R	57	34			
	Taps L	16	6			
	Av Amt R	900 c.c.	950 c.c.			
	Av Amt L	700 c.c.	900 c.c.			

failure before death, and of these one was the case of unilateral left hydrothorax, while the remainder showed fluid bilaterally, with that on the right predominating in five and that on the left in two. In six cases in which pulmonary infarction accompanied the hydrothorax the latter was found to be bilateral and predominantly right in all. Four of this group had had auricular fibrillation, and in all these the hydrothorax was also bilateral with the greater amount of fluid on the right side.

In 23 cases of hypertensive heart disease unilateral hydrothorax occurred only in two instances, once on each side, while bilateral hydrothorax occurred 21 times, in greater degree on the right in 18 and on the left in three. The right pleural space was completely obliterated by fibrous adhesions in the one case showing fluid on the left side alone. Signs of left heart failure had been the outstanding clinical features in 12 of the 23, and all these, except the case of left hydrothorax, presented fluid bilaterally with the greater amount in the right pleural cavity in nine, and in the left in two. In three cases where pulmonary infarction occurred the hydrothorax was bilateral and predominantly right sided in all. Auricular fibrillation had been present in four cases, and the hydrothorax was bilateral in all these, with that on the right greater in three.

Seven cases of syphilitic heart disease presented pure right hydrothorax in one, and in the other six it was bilateral, and predominantly right in five. Left heart failure had been outstanding in five of this group with one showing unilateral right hydrothorax and four showing fluid bilaterally with the greater amount on the right in three. In two cases evidencing pulmonary infarction the hydrothorax was bilateral and greater on the right side.

Because left heart failure had been clinically prominent in a relatively high percentage of the 55 cases, including the coronary, hypertensive, and syphilitic heart disease groups, these were conveniently considered together in calculating the average amount of fluid found in the two pleural sacs. Here again it was observed that the average amount of fluid found in the right sac (550 c.c.) exceeded that found in the left (400 c.c.). Likewise, 26 of these 55 cases had had 40 clinical paracenteses performed, 34 on the right chest and six on the left chest, with the removal of an average amount of 950 c.c. fluid from the right pleural cavity and 900 c.c. from the left.

This entire group of 110 cases of congestive heart failure, studied post mortem, presented hydrothorax on the right side alone in six instances, on the left side alone in five, and bilaterally in 99 or 90 per cent of cases. Of the latter there was found a greater amount of fluid in the right cavity in 85, more in the left cavity in 11 and in three it was equal. Complete obliteration of the right pleural space by fibrous adhesions could account for three of the five cases of pure left hydrothorax. It was thought that an extensive fibrous pleuritis, especially involving the lower portion of these membranes, might have influenced the distribution of the accompanying hydrothorax in 19 other instances, with involvement of the right pleura nine times, of the left three times, and of both right and left seven times. Only in rare instances did

such extensive pleural involvement suggest its presence either clinically or radiologically. Left heart failure had dominated the clinical picture in 31 cases. Among these the hydrothorax was unilateral and right in one instance, unilateral and left in three, and in the remaining 27 it was bilateral with the greater degree on the right in 20, on the left in five and in two it was equal. Recent pulmonary infarction had occurred in 36 cases, often presenting multiple areas, either unilateral or bilateral. In this group hydrothorax occurred on the right side alone in one instance, on the left alone in three, and bilaterally in 32 with a greater amount of fluid found in the right cavity in 31. In only rare instances did any close relation between the occurrence or distribution of hydrothorax and the presence of an infarctive process suggest itself. However, owing to the nature of the fluid, such cases might easily have been excluded from this study. In 1935 Joly [2] gave careful attention to this question and felt that pulmonary infarction seldom causes the formation of an extensive cardiac hydrothorax. Auricular fibrillation had been present in 46 cases, of which hydrothorax was limited to the right side in one, to the left side in two, while in 43 it was bilateral and predominantly right in 40. Although the percentage of occurrence of bilateral hydrothorax in this study was quite high, it would appear that the preponderant involvement of the right pleura was consistently evident regardless of the manner in which the cases were grouped for consideration.

The present observations and studies have led to findings which strongly support the general opinion in regard to the distribution of hydrothorax in congestive heart failure. Thus, when an analysis based on the incidence of thoracenteses was made, a marked tendency for the right chest to predominate the picture was encountered (table 1). In this series of cases one side of the chest was aspirated alone in 91 instances, 88 per cent being performed on the right thorax. When the hypertensive patients were considered as a group, the right thorax was aspirated in 71 per cent of instances. While this tendency was maintained, regardless of the underlying heart condition, it would appear that auricular fibrillation and other factors incident to rheumatic heart disease further favor a preponderance of right hydrothorax. In the second series of cases (table 2) as viewed by the roentgenologist, a greater amount of fluid was observed in the right pleural cavity in 56 per cent of instances and in 27 per cent it appeared to be about equal on the two sides. Although the number of cases considered here was quite small, there again appeared augmented influences favoring right hydrothorax in the rheumatic group. Published statistics on the distribution of hydrothorax, as determined at postmortem examination, appear to be in close agreement; however, the present series (table 3) indicated a greater percentage of bilateral hydrothorax than had been previously reported. This discrepancy may be partially accounted for by the manner of screening the subjects and by the use of different methods of recovering and considering the fluid. In 107 of this series of cases the amount of fluid removed from the right pleural cavity exceeded that removed from the left in 85 per cent

This ratio varied between etiological groups, being found highest in the rheumatic and lowest in the syphilitic, but in no instance did it fall below 80 per cent. These studies indicate that from whatever angle an analysis is attempted, regardless of the method of approach or the underlying cardiac condition, the combination of factors which determine the transudation of fluid into the pleural sacs exerts an influence in such a manner as predominantly to involve the right pleura. This influence, however, appears to become augmented in rheumatic heart conditions as compared to those in which failure of the left heart is more frequently encountered.

Discussion

When one attempts to review the numerous efforts that have been made to explain the distribution of hydrothorax which occurs in the course of congestive heart failure, one is struck by the fact that the subject has been approached from isolated points of view. This has led to an over-emphasis of some particular factor involved and a tendency toward over-simplification of the problem as a whole. Much of the work published has been based on clinical observation of the condition with theoretical conclusions drawn as to the most likely factor determining the distribution of the fluid. In 1867 Bacelli [3] advanced the azygos theory to explain the comparative frequency of right hydrothorax, assuming that pleural fluid accumulated through disturbance in the systemic circulation. His views seem to have been accepted without question until the turn of the century and indeed have made themselves felt until much more recently. In 1904 Steele,[4] reviewing his earlier work (1896) and that of Stengel,[5] reported the occurrence of right hydrothorax alone, or as greater than left, in 60 per cent of clinical cases and in 77 per cent of those observed at postmortem examination. He and Stengel felt that pressure on the root of the right lung and azygos vein by an enlarged right auricle could explain the predominance of right hydrothorax and suggested that the site of a hydrothorax was associated with a corresponding enlargement of the right or left side of the heart.

Crediting West [6] with having expressed similar views previously, Fetterhoff and Landis [7] in 1909 presented a convincing argument that transudation of fluid into the pleural sacs took place from the visceral rather than the parietal layer and therefore depended upon involvement of the pulmonary instead of the systemic circulation. They argued that pressure on the right or left pulmonary veins, by a dilated right or left auricle respectively, determined the location of the pleural transudate. Since the right auricle was more easily and consequently more commonly dilated than the left, a predominance of right hydrothorax might be expected.

More recently (1930) Satke [8] presented experimental data demonstrating a greater relative degree of pressure negativity in the right pleural space, as compared to that in the left, in normal individuals, and suggested this difference would explain the prevailing tendency toward preponderance of

right hydrothorax. This tendency was supported by Famulari [9] on the basis of the anatomical relations existing between the thoracic aorta and the hemiazygos vein and the presence of valves in the latter. Dock [10] in 1935 presented convincing evidence that the anatomic and hydrostatic factors relating to the flow of blood from the pulmonary venous bed to the left ventricle strongly favored the predominance of right over left hydrothorax. He pointed out these factors as being considerably augmented by the right lateral decubital position which cardiac patients generally prefer, according to the studies on "trepopnea" by Wood, Wolferth and Terrell.[11]

Fishberg [12] states that although cardiac hydrothorax is often unequal and usually right sided, no adequate explanation has been given to account for this distribution. He expresses the opinion that transudation into the pleural sacs, due to heart failure, depends upon disturbance of the systemic as well as the pulmonic circulation. In studying left heart failure, Bedford [13] found left hydrothorax in 18 of 38 cases, whereas fluid occurred on the right side alone in only nine instances. Because of the unusual incidence of unilateral left hydrothorax in this series, in opposition to accepted views, Bedford and Lovibond (1941) did a follow-up study including all types of congestive heart failure.[1] They agreed with Steele's earlier idea that there existed a definite relation between the underlying heart condition and the site of the hydrothorax and reached conclusions to which reference has already been made. It is of interest that Weiss [14] in his studies of pulmonary edema found that usually congestion and edema started in the right lung and remained more intense on this side than on the left.

The relative rôles played by the systemic and pulmonary circulations in the pathogenesis of cardiac hydrothorax has been a subject of much speculation and controversy. With the accumulation of clinical and experimental data, however, there seems little doubt that the visceral pleura is to be considered the source of such fluid collections. Graham [15] in 1921, working on the edematous lung excised immediately post mortem, was able to demonstrate the transudation of fluid from the visceral pleura by varying the degree of pressure negativity within the range of normal. He was convinced that the increased pressure negativity produced by forcible inspiration could suck excess fluid through the surface of the lung. Zdansky [16] expressed the view in 1929 that, on the basis of radiological evidence, hydrothorax should be considered as the sequence of pulmonary engorgement and edema. Extensive observations were reported by Yamada [17] in 1933 on several hundred presumably healthy Japanese soldiers, in whom pleural fluid could be aspirated in 29 per cent, and following severe exercise in 70 per cent of the same group. One wonders whether a heightened negative intrathoracic pressure, acting alone, could account for such unusual findings. The amounts of fluid dealt with were too small to warrant an opinion as to its actual distribution.

That almost the entire capillary venous return from the visceral pleura is received by the pulmonary veins has been shown by Miller [18] in 1937.

More recently Drinker,[19] in his noted lectures on pulmonary edema and inflammation, tersely stated, "It is generally acknowledged that two factors are fundamental in causing transudation in the lungs and pleural sacs. They are, first, sustained increase in pulmonary pressure, and second, anoxia—while one or the other may be dominant in a given case, they never, in my opinion, work alone." After discussing the variation, from tissue to tissue, in increased capillary permeability due to anoxia and emphasizing the particular vulnerability of the lung capillaries to this and other influences, he further stated, "It is my belief, I cannot say conviction—that simple pulmonary edema and the more serious pulmonary exudations depend more upon alterations in the permeability of the lung capillaries than upon complicated pressure relations in the pulmonary circulation."

Accepting the view that the visceral pleura is the principal source of abnormal collection of fluid within the pleural sacs, and owing to their peculiar environment, their increased susceptibility to anoxia, and the inadequacy of pulmonary lymph flow under stress,[19] that the pulmonary capillaries are particularly vulnerable to the forces promoting transudation, there are yet to be considered a number of anatomical and physiological factors which may determine, modify, or tend to localize such a process.

A number of these factors pertain to the lungs themselves. The right lung is some 10 per cent greater in volume than the left and, considering the extra lobe on the right, the disproportion between the areas of visceral pleura on the two sides is even greater. Diseases of the lungs or pleurae, active or healed, were considered by Zdansky [16] and Weiss [20] to influence the localization of pulmonary edema and therefore its sequelae. Pleural adhesions may influence the accumulation of pleural fluid either positively or negatively, depending upon the extent of the involvement. Christie and Meakins [21] in their studies on intrapleural pressure changes in congestive heart failure, were able to demonstrate marked decrease in distensibility and impairment in elasticity of the lungs. Working along similar lines, Prinzmetal and Kountz [22] considered the occurrence of a vicious circle in the relation of pulmonary congestion to lung ventilation. Any factor then, which tends to limit respiratory excursion, such as hypostasis, hepatic engorgement, cardiac enlargement, etc., tears down the natural defenses against the consequences of local increased capillary transudation.

It is difficult to visualize local pressure effects on the pulmonary venous return by an enlarged right or left auricle. There seems to be no consistent relation between such enlargement and the site of a hydrothorax from the radiologic point of view. Of greater significance may be gross cardiac enlargement resulting in direct compression of lung tissue. It is clear that the onset of cardiac arrhythmia often initiates congestive failure, but the high incidence of right hydrothorax in the presence of auricular fibrillation, as brought out by Bedford and Lovibond, warrants further study. The pulmonary lymphatic drainage, as shown experimentally by Warren, Peterson and Drinker,[23] takes place almost entirely through the right lymphatic

duct with limited anastomosis to the thoracic duct. One wonders what effect this might have on the lungs individually when lymphatic stasis occurs and whether the left lung receives greater benefit from the collateral drainage. The elective position patients assume, as emphasized by Dock [24] and by Wood et al., further influences capillary leakage and lymphatic stasis as does also the anatomic and hydrostatic factors described by Dock. That a single factor, such as thrombosis of a blood vessel or obliteration of a pleural space, can explain the location of a pleural transudate is clearly understood, but such instances are relatively rare.

It is much more difficult to form an impression as to the relative importance of the parts played by the visceral and parietal pleurae in absorption of fluid, and little experimental work seems to have been done on which to base an opinion. That the visceral pleura is active in the absorption of fluid is apparent in instances of localized interlobar pleural effusions. It is reasonable to regard the mechanism of pleural fluid formation as one in which there is constantly a transudation and reabsorption of fluid in the pleural sac. When excessive amounts are present either or both factors may be disturbed and recovery take place when the normal balance is reëstablished. In consideration of the problem it would seem advisable to keep the fundamental factors of transudation in the lungs and pleural sacs in mind and to realize that in a given case a number of influences may be active together in determining the site of fluid accumulation.

Summary and Conclusions

1. The distribution of hydrothorax in congestive heart failure was determined in three groups of patients by three methods respectively, i.e., by thoracentesis, by radioscopy, and at autopsy.

2. The findings obtained from these three analyses were in close general agreement throughout the study.

3. Depending on the method considered, right hydrothorax predominated in from 56 to 80 per cent of cases and left hydrothorax in from 12 to 17 per cent. Fluid was equally distributed in 3 to 27 per cent.

4. When etiological groups of heart disease were considered, the predominance of right hydrothorax over left was maintained regardless of the underlying heart condition.

5. Rheumatic heart disease and auricular fibrillation appeared to augment the influences determining a right hydrothorax, while pure left heart failure tended to mitigate these to a limited degree.

6. Any explanation for the distribution of hydrothorax in congestive heart failure may be attempted only through consideration of a number of influencing factors. However, it is clear that the balance of these forces is exerted in such a manner as greatly to favor the involvement of the right pleural sac.

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