THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE AND ITS CLINICAL SIGNIFICANCE [1]

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The mechanism of cardiac dyspnoea remains a matter for conjecture although many hypotheses have been advanced. Most of these resolve themselves into a statement that a correlation between dyspnoea and some other physical sign exists. Reduction in the volume of the vital capacity, increase in the venous pressure, reduction in the cardiac output, and so on, have been suggested as the "cause" of cardiac dyspnoea, since they are usually proportional to the degree of respiratory distress. Most, if not all, of those hypotheses which blame impairment of one or other of the mechanisms which are known to control breathing, do not bear the test of experimental analysis. We will review in brief the various theories which have been advanced to describe the cause of cardiac dyspnoea.

Cardiac dyspnoea can conveniently be divided into four groups which are quite distinct clinically, and which presumably are caused by different mechanisms. These are orthopnoea, paroxysmal cardiac dyspnoea (cardiac asthma), Cheyne-Stokes respiration, and lastly dyspnoea on exertion, which, as heart failure with congestion progresses, becomes continuous dyspnoea exaggerated by exertion. The last type is a cardinal symptom of heart failure and the various theories which have been advanced as to its causation will be dealt with first.

a. Imperfect aeration of the respiratory centre, due either to diminished circulation rate of flow or to deficient aeration of the arterial blood, has long been emphasized as one of the primary causes of *cardiac dyspnoea.* Yet there is seldom any significant oxygen unsaturation of the arterial blood and the pressure of CO_2 is almost invariably below normal. The circulation rate is diminished in heart failure with congestion but the evidence is against any significant increase in the tension of CO_2 or decrease in the tension of O_2 in the respiratory centre, although it is possible that in extreme cases such changes may be of minor importance (Meakins and Davies (1925), Fraser (1927), Calhoun, Cullen, Harrison, Wilkins and Tims (1931), and Harrison, Harrison, Calhoun and Marsh (1932)).

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b. Increased ventilation is frequently included among the " causes " of cardiac dyspnoea. Since there is no signicant increase in the basal metabolic rate, the increase in ventilation, which runs pari passu with the degree of respiratory distress, is a sign rather than a cause of cardiac dyspnoea (Peabody, Wentworth and Barker (1917), Harrison, Harris and Calhoun (1931), Harrison, Harrison, Calhoun and Marsh (1932)).

c. Increased venous pressure has recently been suggested as the cause of cardiac dyspnoea (Harrison, Harrison, Calhoun and Marsh (1932)). " Reflex stimulation of respiration because of increased pressure in the right side of the heart and in the cardiac ends of the great veins " is regarded as of especial importance in the genesis of cardiac dyspnoea. The evidence is based on the occurrence of rapid breathing in dogs due to two procedures, both of which raise the pressure in the right auricle. This twofold response can be obtained by injecting suddenly a large quantity of fluid into the venous system. Although rapid breathing results, there are many other systemic changes which occur besides a rise in the venous pressure. Cloetta and Stäubli (1919) have shown that the pressure in the pulmonary artery also rises under these circumstances and that, if the pleural cavities be open to the atmosphere, the volume of the lungs increases, or if this be prevented the functional residual air decreases. This can only mean an engorgement of the pulmonary capillaries resulting in the " Lungenstarre " of von Basch (vide infra). The same rigidity of the lungs is always found in congestive heart failure and can be produced by many experimental procedures such as multiple pulmonary emboli and anaphylaxis, which also result in rapid and shallow breathing and a rise in venous pressure. The relationship of pulmonary rigidity to cardiac dyspnoea will be discussed in greater detail below. The second method employed by Harrison, Harrison, Calhoun, and Marsh, was the inflation of a rubber balloon inserted into the right auricle of a dog. It was necessary to inject over 15 cc. of air into the balloon to obtain a significant increase in the respiratory rate. But they found that even passive movement of a leg may lead by a reflex mechanism to marked respiratory acceleration. The pain of coronary spasm is proverbial and the possibility that the reflex respiratory acceleration which they observed after such an insult to the auricular wall was due to a pain or shock reflex, should be considered.

d. The diminution of the vital capacity has long been known to be approximately proportional to the degree of respiratory distress. All the evidence indicates that diminution is due not to reflex inhibition of inspiration but to decreased distensibility of the lungs (Romanoff (1911), Meakins and Davies (1925), Hofbauer (1925)). No case in which the volume of the vital capacity did not exceed the limits of a normal respiration has been reported; the loose statement, that diminution in the vital capacity is a cause of cardiac dyspnoea, should cease to be repeated. The decreased distensibility of the lungs may increase the amount of work which must

be done to yield a breath of ordinary depth, or in some way may change the sensitivity of the Hering Breuer reflex, but these changes cannot be ascribed to a diminution of the volume of the vital capacity. The relationship of pulmonary distensibility to the Hering Breuer reflex and cardiac dyspnoea will be discussed later.

Orthopnoea has been ascribed to the same factors as have been described in the case of cardiac dyspnoea (Calhoun, Cullen, Harrison, Wilkins and Tims (1931), Weiss and Robb (1933)). It is evident that changes in aeration of the respiratory centre cannot be responsible for orthopnoea. Weiss and Robb conclude that " changes in vital capacity and lung volume are to be regarded as the most important causes of orthopnoea." In other words, orthopnoea is in some way linked with the distensibility of the lung, which has long been known to vary with posture. The relationship of pulmonary distensibility to orthopnoea will be discussed later in greater detail.

Paroxysmal cardiac dyspnoea (cardiac asthma) is closely associated with signs of acute pulmonary congestion and is evidently also caused by a sudden decrease in pulmonary distensibility, from the intense engorgement of the pulmonary capillary bed (Weiss and Robb (1933)).

Cheyne-Stokes breathing is presumably of central origin (Meakins and Davies (1925); Anthony, Cohn and Steele (1932)) and will not be considered further in this communication.

From the evidence presented in this brief review it is evident that changes in distensibility of the lungs are probably of prime importance in the causation of cardiac dyspnoea, orthopnoea and cardiac asthma. The elastic properties of the lungs in heart failure have never been subjected to experimental analysis. In vivo, they have not been measured, and the validity of postmortem observations on the distensibility of the lungs is open to question (Christie and McIntosh (1934)). The methods employed in this investigation have already been fully described (Christie and McIntosh (1934), Christie (1934)).

RESULTS

It has been shown (Christie and McIntosh (1934)) that the elastic properties of a lung can be defined in considerable detail by simultaneous registration of the tidal air and intrapleural pressure. The elasticity of the normal lung is very nearly perfect and its distensibility falls within a comparatively narrow range. An attempt has been made to obtain a record of the intrapleural pressure in seven cases of congestive heart failure. In two, free pressure fluctuation from the pleural space was not obtained. The significance of the failure in these two cases will be discussed later, but in the other five, tracings which cannot be considered normal were secured.

I. Pulmonary distensibility

Instead of negative intrapleural pressure throughout the respiratory cycle, the pressure rises at the end of expiration to that of the atmosphere, or higher (Figures I to VII and Figure IX). Any measure of distensibility involves the force applied to distend the lungs. In cardiac failure it is evident that some of the distension during inspiration is consequent

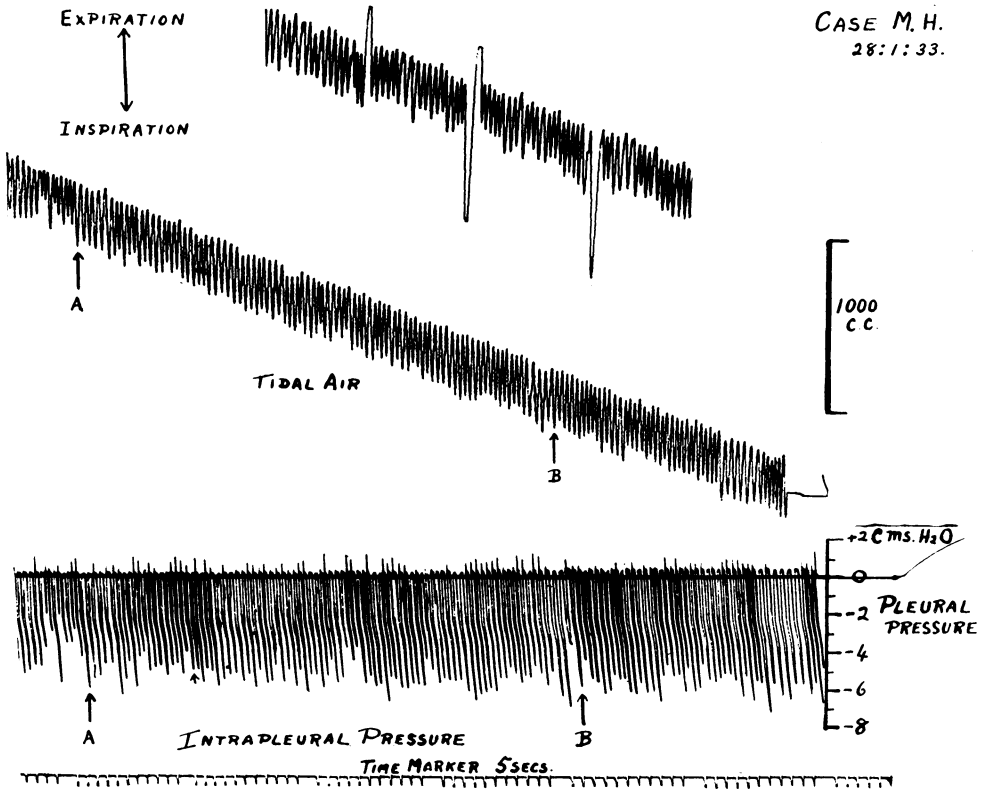

FIGURE I. THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE

Case M. H. The upper tracing is of the vital capacity, and complemental and reserve air taken separately. The middle tracing is of the tidal air taken synchronously with the intrapleural pressure (lower tracing).

on the release from positive pressure applied during expiration. This part reflects the compressibility of the lungs rather than the distensibility. Even in the case of a sponge of perfect elasticity, compressibility, if expressed quantitatively, may be very different from distensibility. Unfortunately, it is impossible to measure the compressibility of normal lungs, since in health they always deflate by elastic recoil; but in a subsequent communication on artificial pneumothorax, we will show that, with the lung partially

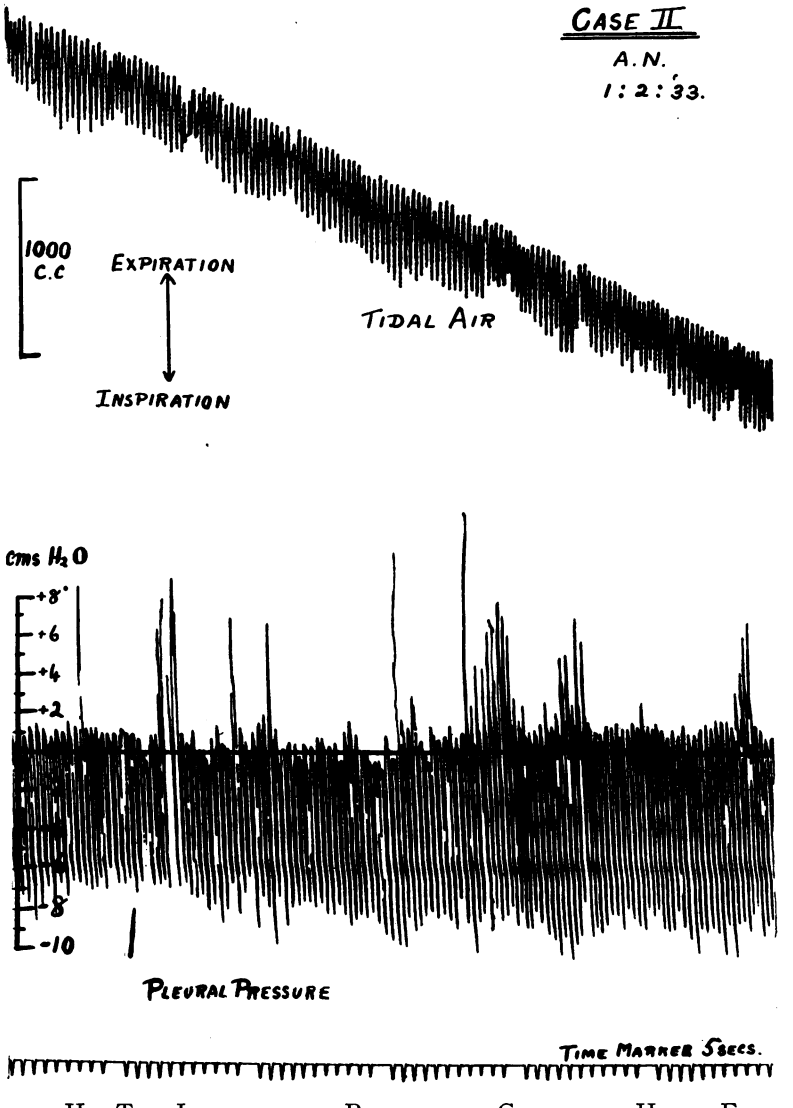

FIGURE II. THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE

Case A. N. The upper tracing is of the tidal air taken synchronously with the intrapleural pressure (lower tracing). The expiratory irregularities in the intrapleural pressure are due to coughing and grunting.

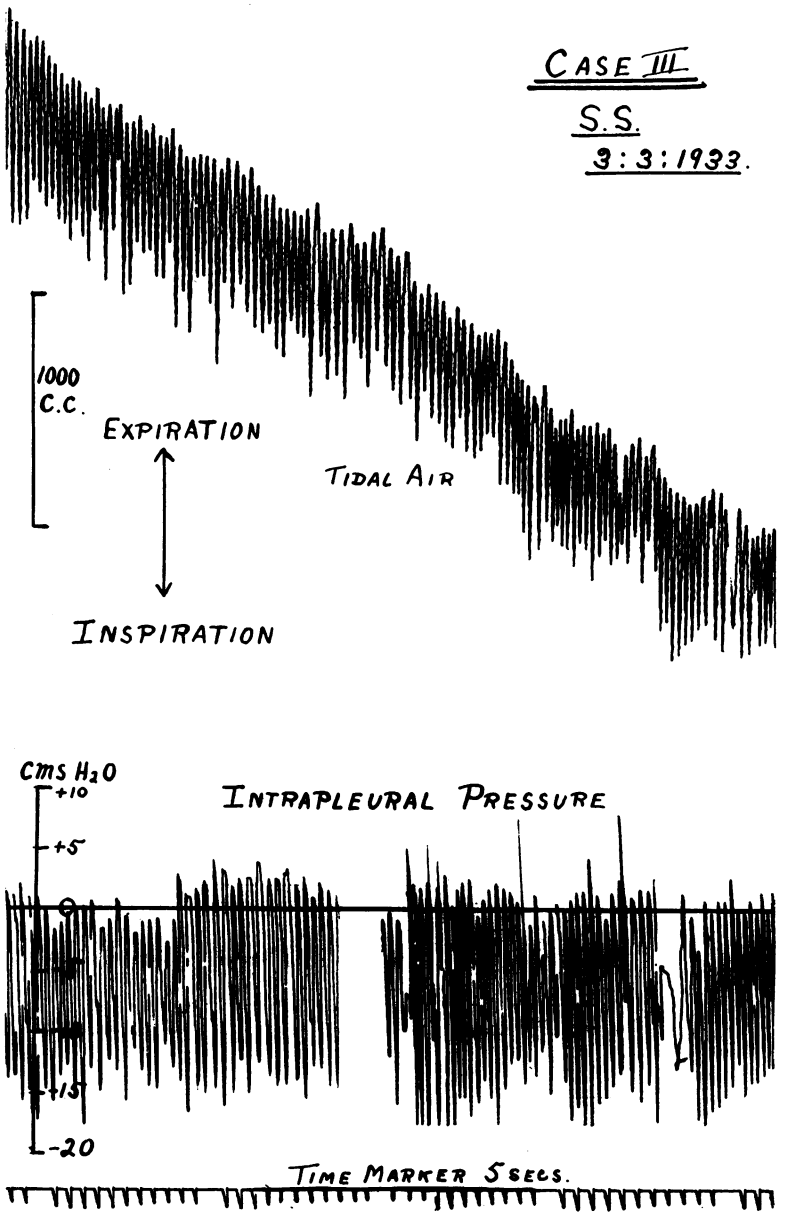

FIGURE III. THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE

Case S. S. The upper tracing is of the tidal air taken synchronously with the intrapleural pressure (lower tracing).

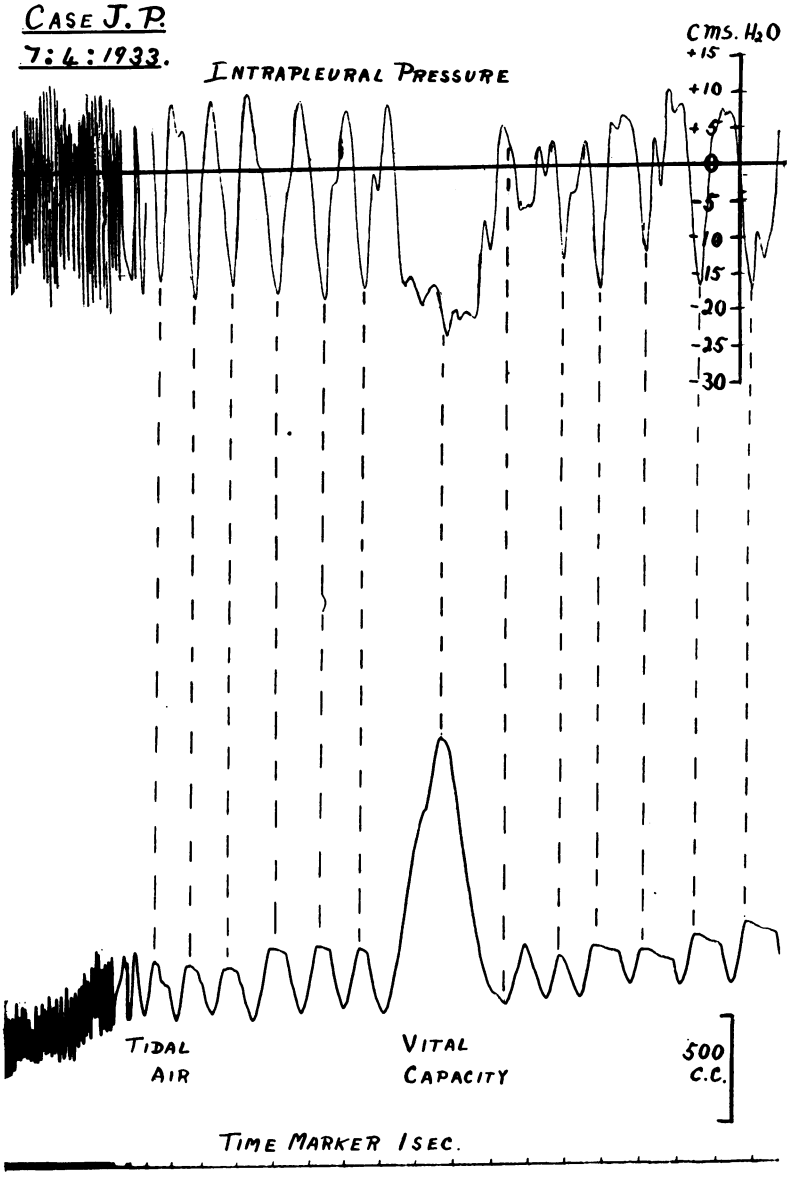

FIGURE IV. THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE

Case J. P. The lower tracing is of the tidal air taken synchronously with the intrapleural pressure (upper tracing). With a fast moving drum the correspondence between the maximum degree of distension when a deep breath is taken, and the point of greatest negativity in the intrapleural pressure is well shown.

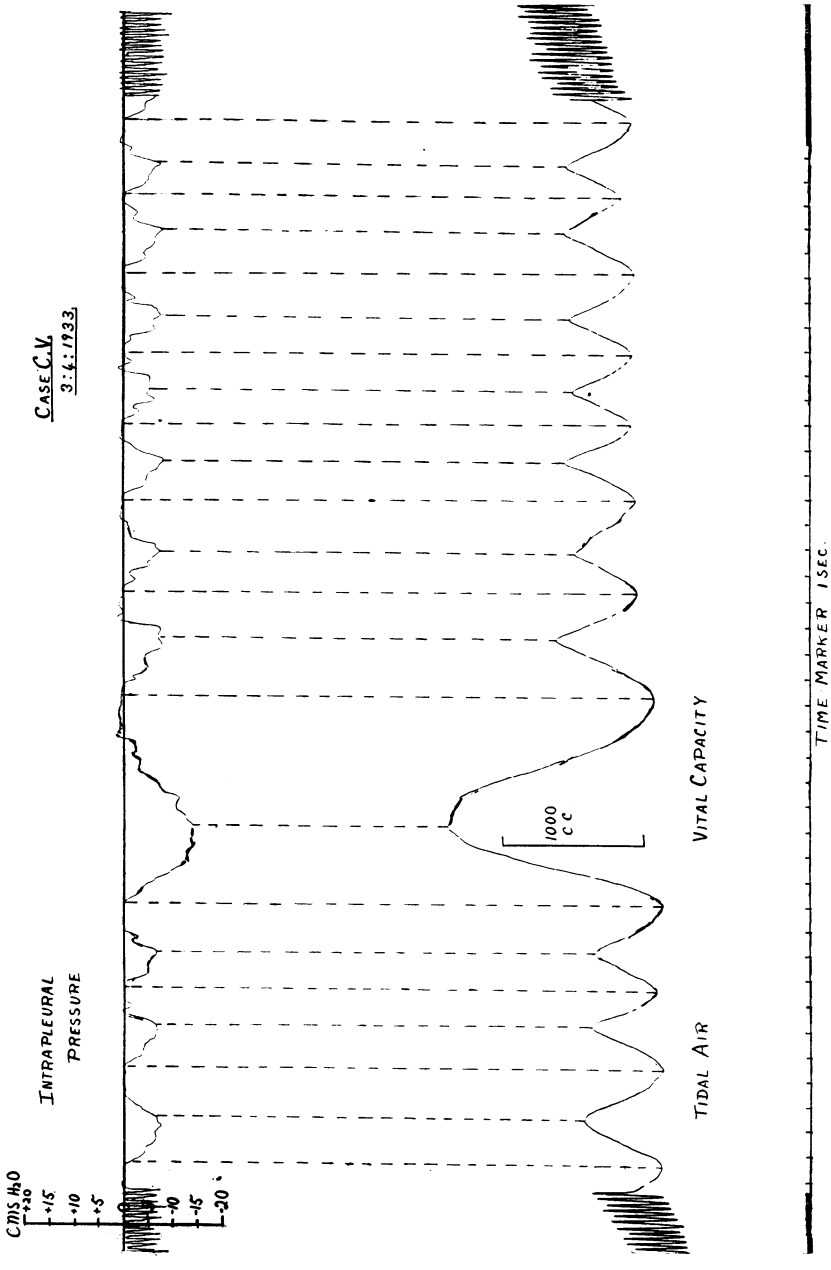

FIGURE V. THE INTRAPLEURAL PRESSURE IN CONGESTIVE HEART FAILURE

Case C. V. The lower tracing is of the tidal air taken synchronously with the intrapleural pressure (upper tracing). With a fast moving drum the correspondence between the maximum degree of distension when a deep breath is taken, and the point of greatest negativity in the intrapleural pressure is well shown.

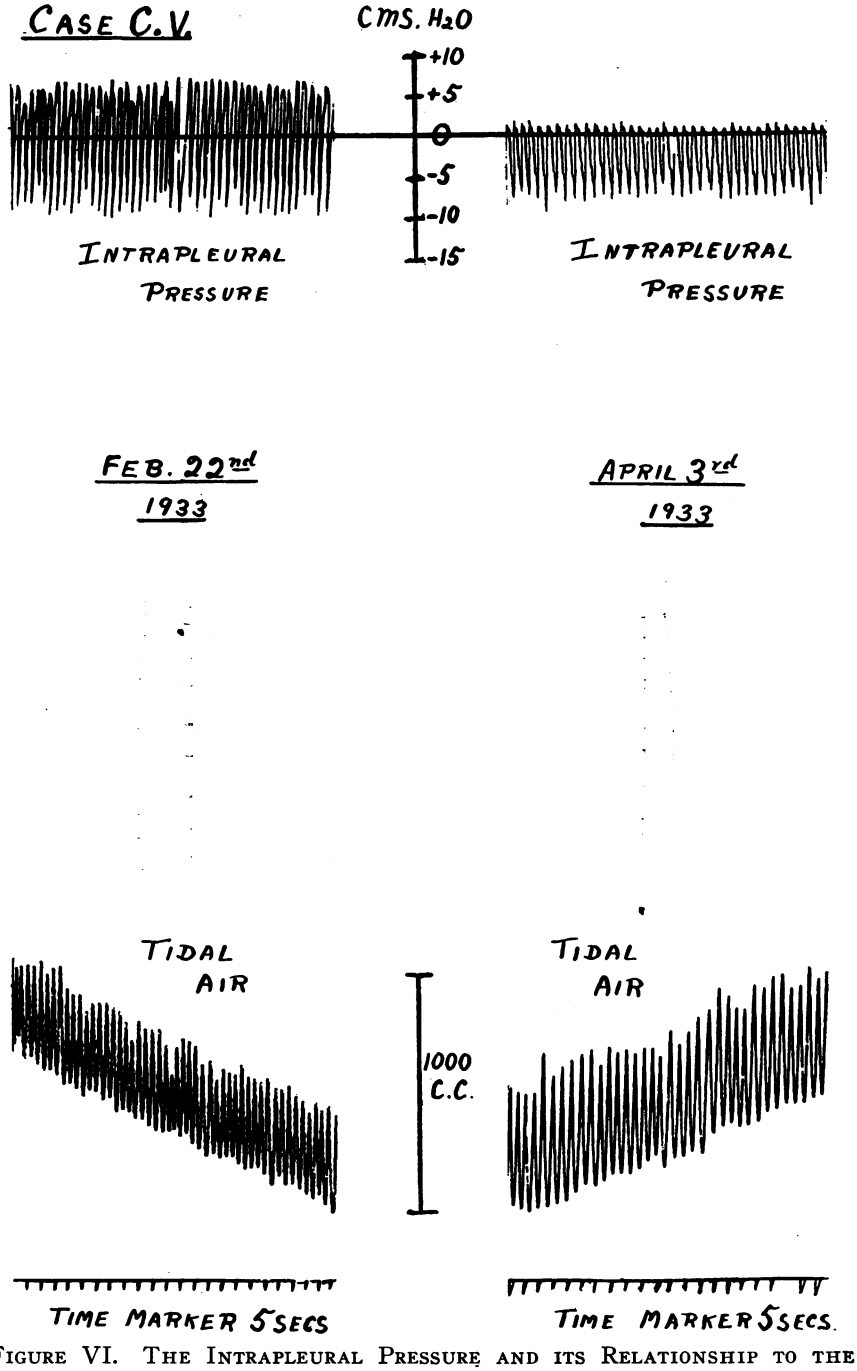

FIGURE VI. THE INTRAPLEURAL PRESSURE AND ITS RELATIONSHIP TO THE DEGREE OF CONGESTIVE HEART FAILURE

Case C. V. After 6 weeks' rest in bed and treatment there is a diminution in the intrapleural pressure fluctuation, even with an increase in the tidal air.

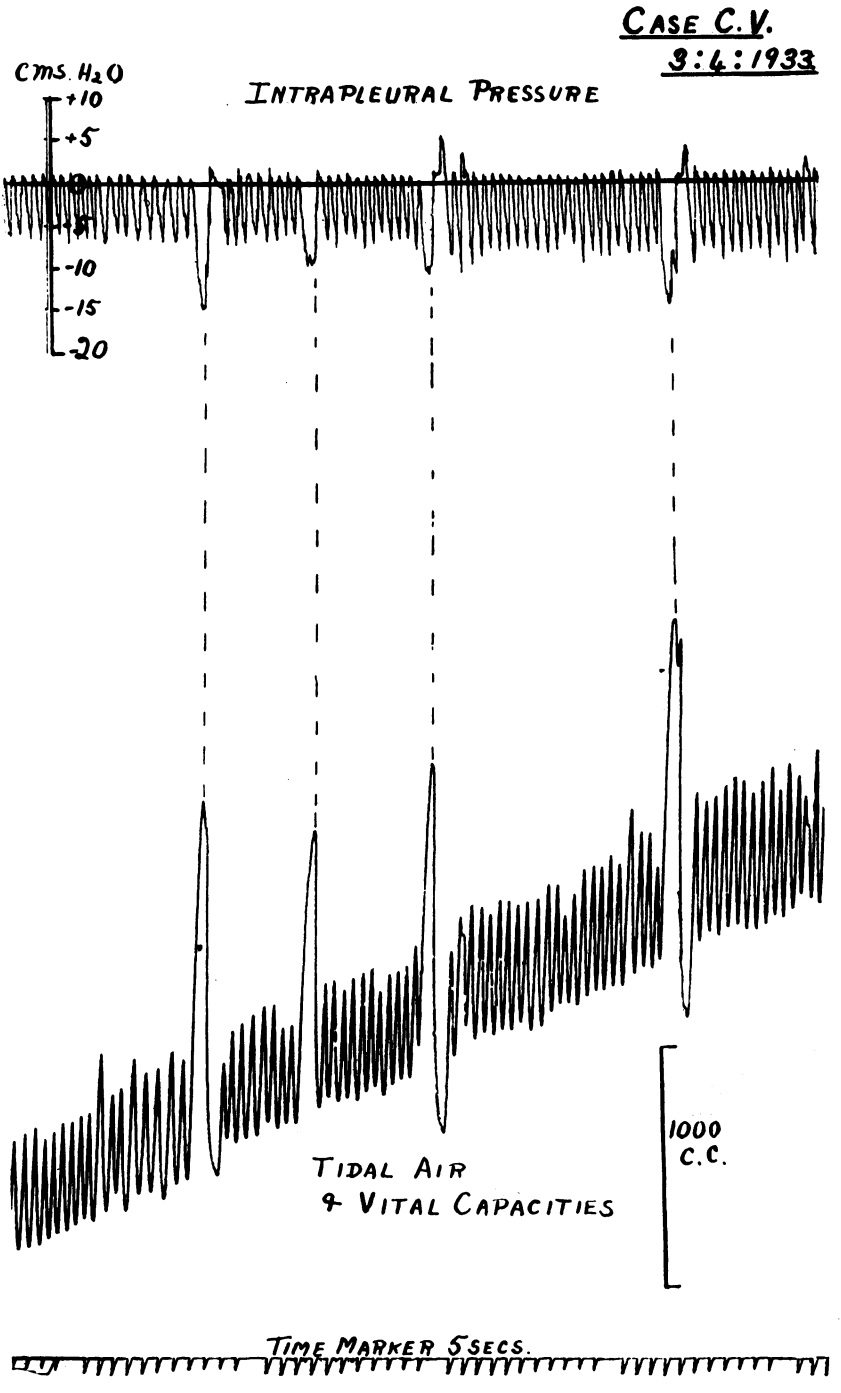

FIGURE VII. THE INTRAPLEURAL PRESSURE AND ITS RELATIONSHIP TO PULMONARY DISTENTION IN CONGESTIVE HEART FAILURE

Case C. V. Any increase in the depth of inspiration is accompanied by a corresponding decrease in the intrapleural pressure.

collapsed, three or four times the force is required to compress the lung as is required to distend it. From this it is obvious that a measure of the distensibility of the lung should only include the negative pressure exerted and only that portion of the inspiration which results from the application of negative pressure. Unfortunately, it is impossible in cardiac failure to distinguish that part of inspiration which is the result of the expanding force. To make this distinction in emphysema is unnecessary, for the intrapleural pressure returns to that of the atmosphere at the end either of a deep inspiration or expiration. Such a loss of elasticity is not evident in heart failure (vide infra). No more is possible than to calculate distensibility on two assumptions; first, that there is no elastic recoil from compression; and second, on the assumption that the compressibility and distensibility are equal and that elastic recoil from compression is perfect. In the former case only the negative pressure exerted on the lungs constitutes the expanding force, while in the latter the total intrapleural pressure fluctuation is included. Both calculations are erroneous; the true figure lies between them (Table I). With either method of calculation the coefficient

TABLE I

Pulmonary distensibility in congestive heart failure

Case	Functional residual air	Distensibility in cm. of H_2O per 100 cc. distension		Coefficient of distensibility *	
		From total pressure exerted	From negative pressure exerted	From total pressure exerted	From negative pressure exerted
	cc.				
S.S.	2905	3.24	2.89	18.8	16.7
M.H.		2.07	1.90		
J.P.	1150	8.78	5.43	20.2	12.5
A.N.	2315	3.09	2.7	14.3	12.5
C.V.					
(February 22, 1933)		3.76	2.21	19.0	11.2
(April 3, 1933)	2530	1.67	1.59	8.45	8.05
Normal				3.5 to 6.0	3.5 to 6.0

* Coefficient of distensibility = The force in cm. of H_2O required to distend the lung by 20 per cent of the functional residual air.

of distensibility is greatly increased. Symptomatic improvement is accompanied by a fall in this coefficient (Case C. V.). The conclusion is drawn from these figures that there is profound impairment of distensibility in the lungs in heart failure with congestion and that this impairment diminishes with symptomatic improvement.

II. Pulmonary elasticity

In Figure VIII the degree of distension is plotted against the force exerted on the lung. In the upper curve this force is taken as the total pressure fluctuation, while with the middle curve only the negative pressure applied is included. The two curves are not parallel but approach one another as the depth of inspiration increases (Figure VIII). The conclusion to be drawn is that release from positive pressure results in a comparatively small amount of pulmonary expansion. The middle curve is then a truer representation of the distensibility than the upper and, although its real position probably lies somewhat higher on the scale, its slope must be representative of the proportionality between stress and strain. It has been shown (Christie and McIntosh (1934)) that in the healthy lung the stress is proportional to the strain but in this case there is a slight increase in distensibility as the depth of breathing increases. This can only mean some impairment of pulmonary elasticity, but an impairment which is small compared with that observed in emphysema (Christie (1934), Figure I). The cruder tests of pulmonary elasticity which have been described (Christie and McIntosh (1934)) fail to reveal any elastic impairment in congestive heart failure. If a deep breath be taken, the point of maximum negativity of the intrapleural pressure corresponds closely to the height of inspiration (Figure IV and V). We have been unable to obtain a record of inspiratory apnoea with the glottis open, but when the vital capacity is measured no " set " can be demonstrated in cases uncomplicated by true emphysema (Figure I). Case S. S. did have signs of mild emphysema. His reserve air averaged 325 cc., when taken alone, and 270 cc., when taken after a deep inspiration. He also shows the increase in functional residual air (Figure IX) and irregularity of the respiratory level (Figure III), which are characteristic of emphysema.

We conclude from these measurements that there is a slight loss of pulmonary elasticity in congestive failure.

III. The lung volume and its subdivisions

There is a decrease in the volume of the vital capacity and a relative increase in the functional residual air (Figures IX and X), (Binger (1923), Meakins and Christie (1929)). The diminution, or even obliteration, of the reserve air is also clearly shown (Hofbauer (1925), Meakins and Christie (1929)). The validity of the lung volume measurements made by Rubow (1908), Bittorf and Forschbach (1910), Siebeck (1912), Peters and Barr (1920) and Lundsgaard and Schierbeck (1923) are open to question and have been discussed elsewhere (Christie (1932)).

IV. Haemo-respiratory exchange

With diminution of the reserve air, which is characteristic of this condition, any measurement of the alveolar air by the Haldane-Priestley

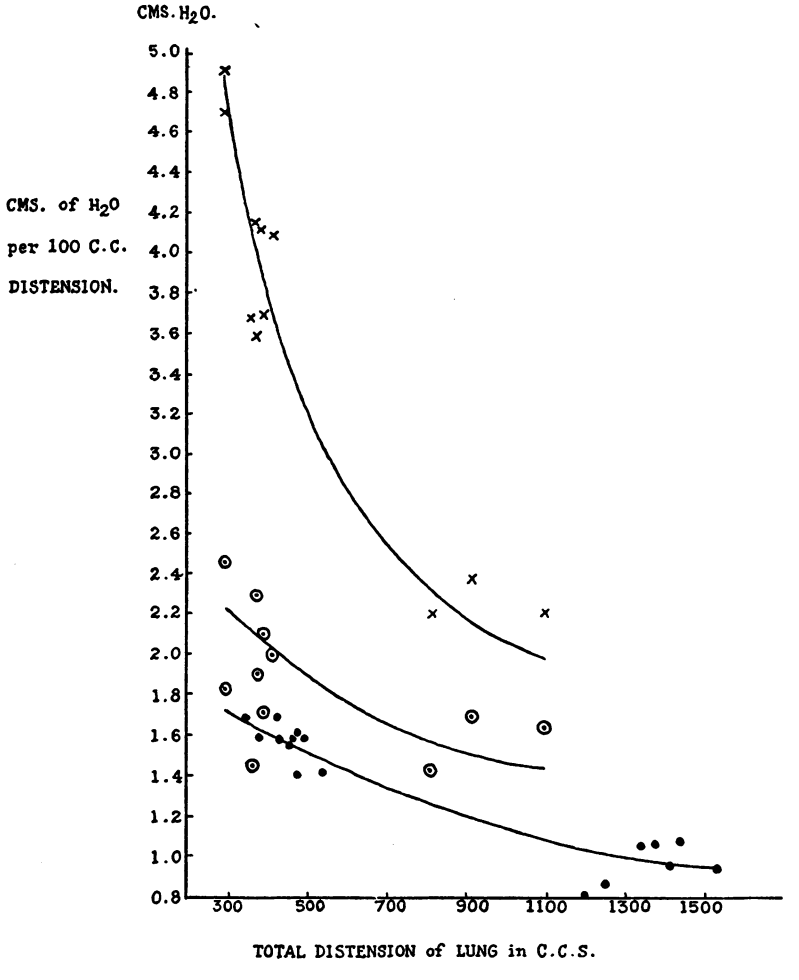

FIGURE VIII. THE PULMONARY DISTENSIBILITY IN CONGESTIVE HEART FAILURE

Case C. V.

X = Depth of inspiration plotted against

$$\frac{\text{Total change in intrapleural pressure}}{\text{Depth of breath in cc.}} \times 100.$$

⊙ and • = Depth of inspiration plotted against

$$\frac{\text{Negative pressure change in intrapleural pressure}}{\text{Depth of breath in cc.}} \times 100.$$

X and ⊙ = From measurements taken on February 22nd, 1933.

• = From measurements taken on April 3rd, 1933, after 6 weeks' rest in bed and treatment.

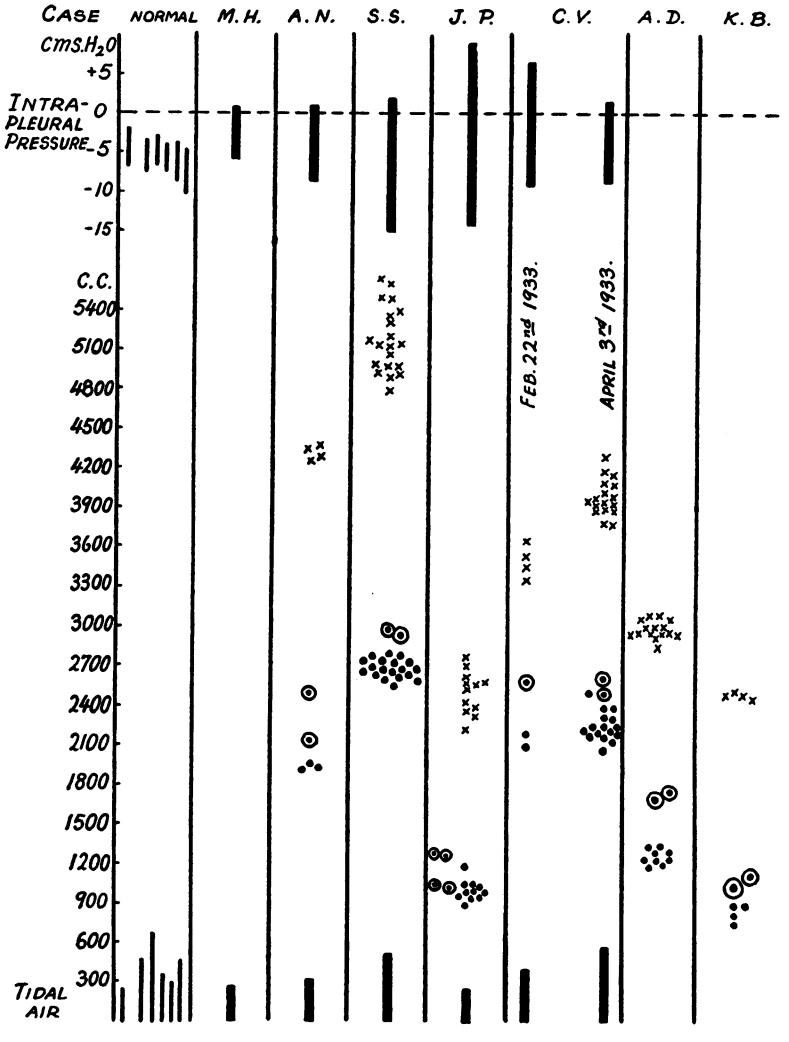

FIGURE IX. THE LUNG VOLUME AND ITS SUBDIVISIONS IN CONGESTIVE HEART FAILURE

X = Total capacity
⊙ = Functional residual air
· = Residual air.

The average intrapleural pressure fluctuation with the corresponding tidal air is shown on 5 cases of congestive failure and 5 individuals with minimal apical tuberculous lesions, with functional residual air ranging from 1300 to 3775 cc.

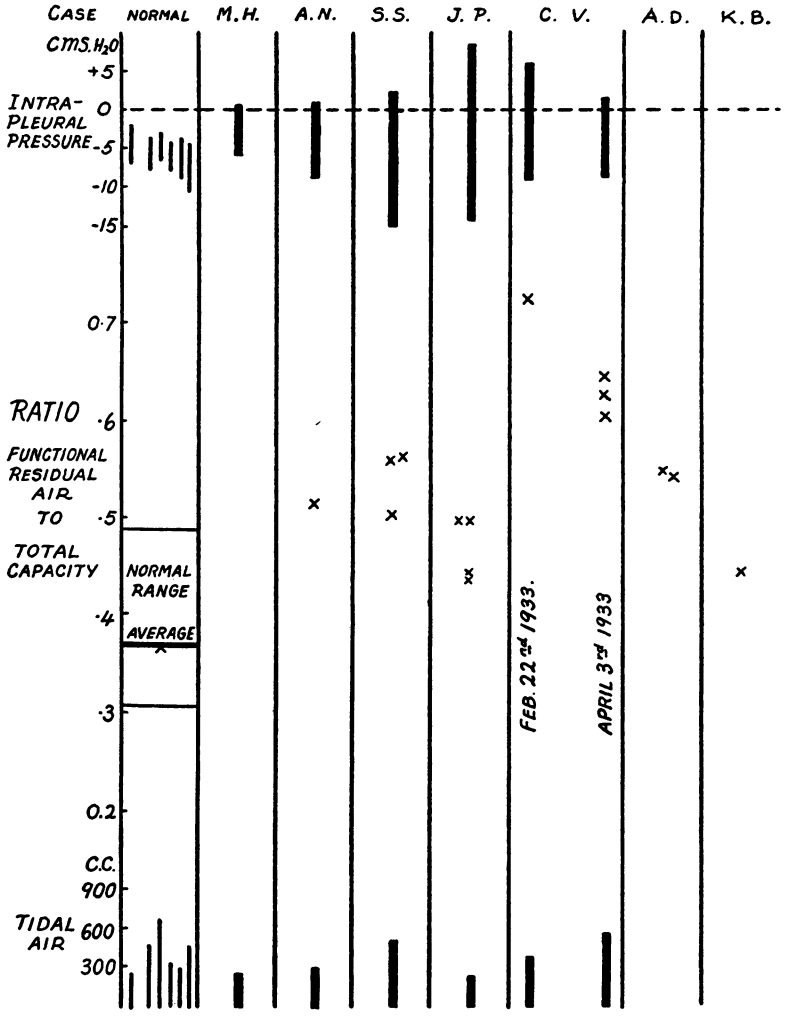

FIGURE X. THE RATIO OF FUNCTIONAL RESIDUAL AIR TO TOTAL CAPACITY IN CONGESTIVE HEART FAILURE

$$X = \text{ratio } \frac{\text{Functional residual air}}{\text{Total capacity}}$$

The average intrapleural pressure fluctuation with the corresponding tidal air is shown as in Figure VIII.

method must be of doubtful significance. What reserve air can be expelled in heart failure is expelled slowly and with difficulty, so that, even if an "inspiratory Haldane-Priestley sample" be taken, the respiratory quotient may be extremely low. It was this type of sample that was secured in Cases A. N., S. S. and C. V. (Table II). The respiratory quotient in all

TABLE II

Haemo-respiratory exchange in congestive heart failure

Case	Tidal air	Respiratory rate per minute	O_2 consumption per minute	Alveolar pCO_2 (Haldane Priestley)	Alveolar pCO_2 (automatic sampler)	Expired pCO_2	Arterial blood		
							O_2 capacity	O_2 saturation	pCO_2
	cc.		*cc.*	*mm. Hg*	*mm. Hg*	*mm. Hg*	*volumes per cent*	*per cent*	*mm. Hg*
A.N.	314	21.2	247	35.5	32.1	19.6	19.75	90.1	36.5
S.S.	687	12.7	344	37.1	28.4	23.6	24.59	94.0	27.0
C.V.	319	15.8	209.5	37.8	29.4	20.0	15.66	89.9	31.0
J.P.	234	23.5	209	26.2	23.3	13.1			
M.H.							21.81	89.5	37.5
A.D.	174	31.2	185.0			18.3	13.66	95.9	36.5

these cases was below 0.6, so that the samples were probably more nearly in equilibrium with the mixed venous blood than with the arterial blood. In Case J. P. it was 0.75 (in the expired air it was 0.83), but with complete absence of reserve air, the sample probably consisted largely of dead space air. When the automatic sampler was used, the respiratory quotient always corresponded with that of the expired air and we believe that, by this method, the pressure of CO_2 reflects more closely the pressure in the alveolar air. Such a supposition is supported by the pressures found in the arterial blood.

The oxygen saturation of the arterial blood was always below normal but in no case was sufficient anoxaemia present to account for the presence of dyspnoea.

In all our cases, including S. S., a case of slight emphysema, the haemo-respiratory picture is typical of congestive heart failure (Meakins and Davies (1925); Fraser (1927); Calhoun, Cullen, Harrison, Wilkins and Tims (1931); and Harrison, Harrison, Calhoun and Marsh (1932)).

DISCUSSION

I. Cardiac dyspnoea

In 1891 von Basch described the rigidity ("Lungenstarre") of the lung in heart failure with congestion. He and his associates showed at autopsy that the lungs were less distensible than normal and suggested that in this mechanical limitation of inspiration lay the cause of cardiac dyspnoea.

This postmortem change has been confirmed by many investigators, some of whom have suggested that, instead of a mechanical limitation of inspiration, some disturbance of the Hering Breuer reflex is responsible for the respiratory embarrassment (Meakins and Davies (1925), Hofbauer (1925), Field and Bock (1925–26), Anthony (1930), Fraser (1927), Harrison, Calhoun, Cullen, Wilkins and Pilcher (1932)). The inference which has been drawn from postmortem observations on the degree of distensibility of the lungs is open to question (Christie and McIntosh (1934)), but we have been able to confirm the observations of von Basch in every respect by measurements made in vivo. Experimental pulmonary congestion (Churchill and Cope (1929)) and other conditions, such as multiple pulmonary embolism (Moore and Binger (1927)), which increase the rigidity of the lung, lead to reflex rapid and shallow breathing which is abolished by vagal section. It is reasonable to suppose that the same mechanism may be responsible for cardiac dyspnoea.

We have already emphasized our belief that all the direct evidence in the literature indicates that cardiac dyspnoea is in some way associated with changes within the lung, rather than with central stimulation. The only demonstrable change in the lungs which can be linked with respiration is decrease in distensibility. It has been suggested that stiffness of the lungs mechanically limits the volume of the tidal air in the same way as it limits that of the vital capacity. In our studies on artificial pneumothorax, where the vital capacity can be slowly reduced to a volume smaller than is found in most cases suffering from cardiac dyspnoea, there has been found no evidence that respiratory distress exists even on mild exercise. There is ample evidence, however, that certain experimental procedures which increase the rigidity of the lung lead reflexly to rapid and shallow breathing. The concensus of opinion still centers on the idea that the Hering Breuer reflex is dependent on change in tension within the lungs. It seems reasonable to suppose that cardiac dyspnoea is largely, if not wholly, dependent on increased sensitivity of this reflex, resulting from the decreased pulmonary distensibility.

Orthopnoea seems in the same way to be linked with some change in the lungs due to posture, though the relation of changes in posture to changes in pulmonary distensibility is less obvious. A simple explanation was given by Hill in 1895, in the course of an inquiry into the effect of gravity on the circulation of the blood (Field and Bock (1925–26)). After having made other observations, which have since become commonplaces, he suggests that the upright position relieves orthopnoea by draining blood into the splanchnic area (and liver). Field and Bock (1925–26) have observed diminution in the rate of blood flow in the upright position confirmed by Bielschowsky and others (Bielschowsky, 1932)) and believe this to be due directly to an effect of gravity in impeding the return of blood to the heart. They discuss the occurrence of stagnation of blood in the

dependent parts, and the consequent relief of pulmonary congestion. The experiments of Hamilton and Morgan (1932) also suggest some such peripheral stagnation and decrease of pulmonary congestion in the upright position. Diminution of pulmonary congestion results in increase in distensibility. If a relation between pulmonary distensibility and the Hering Breuer reflex exists, the mechanism of the relief of orthopnoea becomes comprehensible.

It has been shown (Christie and McIntosh (1934)) that change from the recumbent to the sitting posture results in decrease of intrapleural pressure by some 2 cm. of H_2O. A corresponding increase in the effective venous pressure no doubt results (Henderson and Borringer, see Wiggers (1923)) and in consequence an increase in the output of the right ventricle. The effect of this decrease in the intrapleural pressure must, however, even in health be more than counterbalanced by the hydrostatic decrease in venous pressure at the level of the right auricle. The decrease of intrapleural pressure in assuming the upright position is of further interest, in the light of circulatory adjustment to posture.

Cardiac asthma (paroxysmal cardiac dyspnoea) is difficult of experimental approach, since the factors governing its onset are unknown. There is a supposition that its onset is linked with sudden changes in pulmonary distensibility (Weiss and Robb (1933). The rationale of associating change in distensibility with dyspnoea has already been described.

II. The breathing in congestive heart failure

It is evident that in heart failure expiration is the result partly of positive intrapleural pressure generated by active expiratory effort. In emphysema expiration is due wholly to such an effort (Christie (1934)). In congestive heart failure there is, however, only a moderate impairment of the pulmonary elasticity so that expiration in whole or in part is still the result of elastic recoil. The fact remains that a positive intrapleural pressure exists at the end of expiration, limits the ascent of the diaphragm and impairs in general the efficiency of the respiratory mechanism. The reasons for such a statement have been fully dealt with elsewhere (Christie (1934)). The rationale and efficacy of increasing intra-abdominal pressure by means of an abdominal belt, in the symptomatic relief of emphysema, has also been described (Christie (1934)). Increase in intra-abdominal pressure should be beneficial also in cardiac dyspnoea. In a limited number of cases, so treated, dyspnoea at rest is diminished and patients are capable of greater exertion.

The diminution, or even absence, of the reserve air, now shown to be characteristic of congestive failure, is, as in emphysema, presumably due in part to paradoxical movements of the thoracic cage, and in part to diminished compressibility of the lung. Diminution in the complemental air, in a comparable way, is due to decreased expansibility of the lungs. The

relative increase of the functional residual air can be fully accounted for on the basis of diminished total capacity.

III. Haemo-respiratory exchange

It is generally believed that the anoxemia of heart failure is largely due to impairment of diffusion between blood and alveolar air, caused by the presence of alveolar exudate and thickening of the alveolar walls. Since CO_2 is thirty times as soluble in these fluids as O_2, the excretion of CO_2 is little impaired in comparison with absorption of O_2. This sounds a very reasonable explanation for the low pressure of CO_2 in the arterial blood, and anoxemia, which are characteristic of congestive failure, and in most cases this mechanism is probably of importance. In many milder cases (as A. N. and S. S.) very little exudate can be demonstrated in the lungs and it seems unlikely that mechanical impairment of diffusion can be wholly responsible for the disturbance in haemo-respiratory exchange. Loss of pulmonary elasticity and increased rigidity constitutes another mechanism which may hinder the absorption of oxygen and yet not diminish the excretion of CO_2. A defect in pulmonary elasticity may cause unequal ventilation of the lungs (Christie (1934)), so that the peripheral alveoli are hyperventilated at the expense of those situated more deeply. Decrease in distensibility tends to increase mal-distribution of ventilation, and it seems reasonable to suppose that in congestive heart failure the conditions are such that this should occur, with hyperventilation of some alveoli and hypoventilation of others. If the circulation of the hyperventilated alveoli is normal one would expect anoxemia and an increased excretion of CO_2, since, from the dissociation curves of O_2 and CO_2, the saturation of blood with oxygen cannot be significantly increased, while the excretion of CO_2 can be greatly augmented. Such a mechanism must occur in congestive failure and the relative importance between it and the presence of alveolar exudate must vary from case to case.

IV. The venous return to the heart

The flow of blood especially from the inferior vena cava to the heart is largely governed by the suction of negative intrathoracic pressure. A small but definite pressure gradient exists from the abdominal veins to the opening of the vena cava into the right auricle, where the pressure is lowest (Eyster (1929)). In heart failure the intrathoracic pressure is actually positive during a large part of the respiratory cycle and offers considerable obstruction to the return of venous blood. This probably accounts in part for the rise in peripheral venous pressure and may be of paramount importance in the diminution of the " effective venous pressure " and cardiac output (Wiggers (1923)). It may well be, that part of the symptomatic improvement observed both in heart failure and in emphysema, with the

use of an abdominal belt, is due to facilitation of venous return by the increase in intraabdominal pressure.

V. Emphysema in congestive heart failure

With the decrease of distensibility which has been demonstrated in congestive heart failure, the alveoli are subjected to increased tension change with inspiration and expiration. It is not unreasonable to suppose that these surface alveoli might readily be overstretched and suffer a further loss of elasticity with the production of emphysema. The high incidence of emphysema in cases with chronic congestion has frequently been noted. It is this mechanism, so we believe, which leads to the emphysema of high altitudes. There is ample evidence in the literature to show that at high altitudes the lungs are congested and that emphysema develops. In unpublished investigations we have been able to demonstrate that the lungs of rats, acclimatized to a height of 18,000 feet, show a marked decrease in distensibility and that emphysema, mainly at the peripheral portion of the lungs, progressively develops.

SUMMARY AND CONCLUSION

1. From simultaneous tracings of the volume of the tidal air and of intrapleural pressure in heart failure with congestion, a marked decrease in the distensibility and slight impairment of the elasticity of the lung can be demonstrated.

2. Characteristic changes in the volume of the lungs are described; an explanation for these changes is advanced, on the basis of a decrease of pulmonary distensibility.

3. An analysis of haemo-respiratory exchange does not yield an adequate explanation of the functional impairment in this type of heart failure.

4. Evidence is presented, which rests on the basis of impairment of pulmonary distensibility and elasticity, that the peripheral alveoli are overventilated at the expense of the deeper alveoli. The relative importance of this and other mechanisms which occasion anoxemia accompanied by increased excretion of CO_2 is described.

5. Expiration in congestive heart failure is shown to be, in part, the result of positive intrapleural pressure generated by active muscular effort. The functional drawbacks of this type of expiration are emphasized, in particular diminution in the excursion of the diaphragm, with consequent impairment of inspiratory efficiency.

6. An increase of intra-abdominal pressure by means of an abdominal belt is of value in the treatment of heart failure with congestion. The rationale of this form of treatment is described.

7. Cardiac dyspnoea and orthopnoea can both be fully explained, on the basis of increased sensitivity of the Hering Breuer reflex due to changes in the distensibility of the lungs.

8. The significance of increased intrapleural pressure, in its relationship to venous pressure and the return of venous blood to the heart, is emphasized.

9. Decreased pulmonary distensibility, as a causative factor in the production of emphysema, is described, and an explanation for the high incidence of emphysema in heart failure and at high altitudes, advanced.

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APPENDIX

Case histories

M. H.—Female—Aet. 59.

Palpitation and dyspnoea on exertion for 2 years. Slight orthopnoea. Rheumatic fever in childhood. Blood pressure 175/100 with obvious auricular fibrillation. Arteriosclerosis. Heart rate 95 to 110. Transverse diameter of heart 14.5 cm. Heart-thorax ratio 0.59. No valvular disease. Percussion note impaired at bases with numerous moist râles. Liver and spleen not palpable. No oedema.

Diagnosis: Hypertension with auricular fibrillation and early congestive heart failure.

A. N.—Male—Aet. 25.

Palpitation and dyspnoea on exertion progressively getting worse during past 5 years. Scarlet fever in childhood. Obvious mitral stenosis with rough presystolic and soft diastolic and systolic murmurs. Transverse diameter of heart 16.3 cm. Heart-thorax ratio 0.6. X-ray of heart and electrocardiogram typical of mitral stenosis. Occasional moist râle at either base. Liver and spleen 1 finger's breadth below costal margin. No oedema.

Diagnosis: Mitral stenosis and regurgitation with early congestive heart failure.

S. S.—Male—Aet. 44.

Shortness of breath on exertion and swelling of ankles of 3 months' duration. Some orthopnoea for 2 to 3 weeks. Heart markedly enlarged. X-ray—Greatest transverse diameter 21.3 cm. Oblique diameter 21.5 cm. Transverse aorta 7.0 cm. Heart-thorax ratio 0.61. No valvular disease. Blood pressure 210/120. Occasional moist râle at right base. Liver 3 fingers' breadth below costal margin.

Diagnosis: Hypertension and congestive heart failure.

J. P.—Female—Aet. 22.

"Heart Trouble" with breathlessness on exertion since rheumatic fever 7 years ago. Since a miscarriage 8 months ago has had progressively increas-

ing weakness, breathlessness on exertion, orthopnoea and oedema of legs. Now sleeps in chair. Slight cyanosis. Obvious signs of mitral and aortic stenosis and regurgitation with auricular fibrillation. Transverse diameter of heart 19.5 cm. Heart-thorax ratio 0.675. Blood pressure 120/60. Moist râles at either base. Marked ascites and oedema of legs and back. Liver 4 inches below costal margin.

Diagnosis: Chronic myocarditis and endocarditis, mitral and aortic stenosis and regurgitation, auricular fibrillation and very advanced congestive heart failure.

C. V.—Male—Aet. 61.

February 22nd, 1933. Weakness and fatigue for the past 5 years. Dyspnoea on exertion, orthopnoea and swelling of legs, 6 months' duration. Swelling of abdomen 2 weeks' duration. Slight cyanosis. Some arteriosclerosis. Signs of aortic regurgitation with cardiac enlargement. Blood pressure 164/64. Transverse diameter 19.2 cm. Heart-thorax ratio 0.62. Moist râles at either base. Some fluid in both pleural cavities. Liver enlarged and pulsating. Ascites and oedema of back and legs. Blood Wassermann + + +.

April 3rd, 1933. With rest in bed and mild antiluetic and digitalis treatment, patient improved markedly. Oedema, ascites, dyspnoea and general condition much improved.

Diagnosis: Arteriosclerosis, syphilitic aortitis, aortic insufficiency, advanced congestive heart failure.

A. D.—Female—Aet. 38.

Dyspnoea and palpitation on exertion, anginal-like pains and swelling of ankles—2 years' duration. Typical signs of mitral stenosis and regurgitation. Heart 4½ cm. to right, 7½ cm. to left. Blood pressure 148/120. Moist râles at both bases. Liver and spleen 1 to 2 fingers' breadth below the costal margin. No oedema.

Diagnosis: Mitral stenosis and regurgitation with congestive heart failure.

K. B.—Female—Aet. 32.

Palpitation, dizziness and dyspnoea on exertion since delivery of 4th child, 8 months ago. Slight orthopnoea. Questionable cyanosis. Signs typical of auricular fibrillation with mitral stenosis and regurgitation. Blood pressure 95/80. Heart rate 87. Venous pulsation in neck. Transverse diameter of heart 17.4 cm. Heart-thorax ratio 0.7. Moist râles at either base. Liver one hand's breadth below costal margin. Spleen just palpable. No oedema.

Diagnosis: Chronic myocarditis with auricular fibrillation, chronic endocarditis with mitral stenosis and insufficiency, congestive heart failure.