PULMONARY FUNCTION IN CONGESTIVE HEART FAILURE

Part I. A review of the literature.

Part II. An experimental study of the effect of posture on the mechanical properties of the lungs in congestive heart failure.

by

T. Edward Cuddy, M.D.

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Abstract

Part I

A review of the literature up to April 1957 on pulmonary function in congestive heart failure has been undertaken in an attempt to ascertain if measurements of the disturbances in function of the lungs might explain the dyspnoea associated with this condition. In severe failure nearly all measurements indicated disturbance in pulmonary function. Though deviations from normal values were found in mild and moderate congestive failure, there were no consistent abnormalities to help explain the basis of cardiac dyspnoea, nor did they correlate with the degree of clinical disability.

Part II

The mechanical properties of the lungs were measured in the sitting and supine positions in five normal subjects and five patients with orthopnoea due to congestive heart failure. The patients had higher elastic and viscous resistances in the sitting position. Recumbency increased both resistances in both groups but there was a disproportionate increase in viscous resistance of the patients. This increase was most marked in late expiration and early inspiration. It was concluded that orthopnoea is due to recumbency increasing the viscous resistance and that the effect is related to the level of lung inflation.

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Part I. A Review of the Literature

I. Introduction

Respiratory distress or dyspnoea is frequently an early symptom of congestive heart failure. Sir Thomas Lewis said, "The first indication of cardiac failure is to be found in a diminished tolerance of exercise. Of the very numerous tests of cardiac efficiency and inefficiency, based as they are mainly upon pulse-rate or upon blood pressure or upon both, there is none that approach in delicacy the symptom, breathlessness"(1). Though the etiology of dyspnoea may be complex or multiple, dyspnoea is really a manifestation of pulmonary insufficiency (2).

Studies of the alterations of pulmonary function in patients with heart failure might enable one to understand the basis of this distressing symptom and allow assessment of the clinical disability. Therefore, a review of what is known of pulmonary function in heart failure has been conducted.

II. Lung Volume

After the introduction of the spirometer by Hutchison in 1847, Rubow in 1908 (3) and Siebeck in 1910 (4) found a reduced vital capacity in congestive heart failure. This was later confirmed by Peabody and his associates (5) in a series of investigations of respiration in congestive failure conducted at Harvard University in 1916-17. They found vital capacities less than 90% of normal in 131 of 156 patients with heart failure; the vital capacity was less than 60% of normal in severe failure. Recovery from failure was associated with an increase in vital capacity. The vital capacity was then thought to parallel dyspnoea very closely and be a good index of disability.

Further subdivisions of lung volume were measured (6-11) and vital capacity, inspiratory and expiratory reserve volume, residual volume and total lung capacity were all reduced in severe failure. Earlier studies (3) showed a relatively larger residual volume in mild congestive failure which was thought to be associated with a decrease in expiratory reserve volume. However, in severe failure all lung volumes, including residual volume, fell (7,8). This was confirmed by Richards in 1951 (11) who graded lung volumes according to severity of failure in a series of 26 patients with varied etiological cardiac diagnoses. In mild failure there was an elevation of the resting level or mid position with an increased functional residual capacity and residual volume and a decreased inspiratory reserve volume. With increasing degrees of failure there was a fall in all volumes with the decrease in residual air occurring only in frank congestive failure. He did not report differences between left ventricular failure and mitral stenosis.

More recently (12,13) ventilatory studies in mitral stenosis indicated normal lung volumes in compensated cases. When failure occurred there were progressive symmetrical reductions in inspiratory and expiratory reserve volume, and hence vital capacity (13). Residual volume was found to increase during early failure and remain unchanged with severe failure, during which there was a fall in total lung capacity. Thus the ratio of residual volume to total lung capacity rose to abnormal levels. Vital capacity was reduced further in patients with congestive heart failure due to hypertensive disease than in that due to mitral stenosis, according to a study of the two etiological groups of patients with failure of similar severity (14).

The reasons for changes in lung volume are complex. Diminished vital capacity was early thought to be due to 'rigidity' of the lungs (5,7,9,10). Encroachment of the lungs by an increased heart size, pleural effusion or an elevated or restricted diaphragm may also occur (8,9,11). Muscular weakness and fatigue were considered as important factors in lowering the vital capacity (12,15). It has also been suggested that an increase in pulmonary blood volume might cause rigidity of the lungs and encroach upon the ventilatory space (16,18). In fact, procedures designed to increase pulmonary blood volume were shown to lower the vital capacity (19-22) and the pulmonary blood volume was shown to be increased in left ventricular failure and be relatively normal in mitral stenosis (17,18). This might explain the finding of a low residual volume in left ventricular failure (11,14) and a normal residual volume in mitral stenosis.

Correlations of diminished vital capacity and elevated pulmonary vascular pressures were variable. Berglund, 1949, (25) reported a negative correlation between vital capacity and pulmonary capillary pressure in mitral stenosis while Borden et al (26) found the correlation between pulmonary artery pressure and vital capacity to be only fair in mitral stenosis and absent in left ventricular failure.

From the earlier opinion that vital capacity was a good index of disability (5,23), it is now apparent that lung volumes may be normal in the presence of pronounced dyspnoea (9,12,13,15,24) and are only consistently abnormal in marked congestive failure, when all lung volumes are reduced.

III. Dynamic Function (Pulmonary Dynamics)

Since the term 'ventilation' implies the dynamic displacement of the outside atmosphere into the lungs (2), it is not surprising that studies

of the dynamics of this displacement have been helpful in assessment of pulmonary disease (27). We might expect such studies in cardiac disease to yield a similar result.

Resting ventilation has long been known to be elevated in congestive failure (28-31). Similarly, oxygen consumption and the basal metabolic rate were also shown to be increased in this state (28,32). Hyperventilation was suggested by finding an increase in the ventilation per unit of oxygen consumed, or ventilatory equivalent (12,13,29,33), and a low alveolar CO_2 tension (27,34,35). The hyperventilation of cardiac dyspnoea is still not adequately explained. Cullen et al (35) felt that there was no relation to abnormalities of blood gases or acid-base balance. Resnick (32) thought the increased ventilation was due to increased cost of ventilation in terms of O_2 consumption - which has since been shown (12,13,29,33) to be not proportionate. McMichael (33) felt reflex hyperventilation was produced by anoxemia of the respiratory centre due to a diminished cerebral blood flow and low cardiac output, but more recent workers have invoked reflex stimulation from congestion of left atrium, pulmonary veins, capillaries or adjacent tissue (12,27).

In the assessment of disability due to pulmonary disease, considerable use has been made of the maximal breathing capacity and the following derived indices:

1. Air velocity index - $\frac{\%\text{ of predicted M.B.C.}}{\%\text{ of predicted V.C.}}$ (37)

2. Breathing reserve - $\frac{\text{M.B.C. - resting ventilation}}{\text{M.B.C.}} \times 100$ (2)

3. Walking index - $\frac{\text{Walking ventilation}}{\text{M.B.C.}} \times 100$ (38)

In mitral stenosis nearly normal values were found in ambulatory patients with considerable exertional dyspnoea (12,13), and these became abnormal only as congestive heart failure became severe (13). In pulmonary disease dyspnoea was absent until the breathing reserve fell to less than 65% of the maximal breathing capacity, but many cardiacs were dyspnoeic with values of over 75% (27). Similarly, many patients with mitral stenosis became dyspnoeic after less exercise than their 'walking index' would indicate. Timed vital capacity and air velocity index remained normal, indicating symmetrical changes in vital capacity and maximal breathing capacity (13). When the maximal breathing capacity was reduced, it seemed to be a result of diminished distensibility and muscular fatigue (36,39) rather than airway obstruction. Casby et al (14) recently studied ventilatory function in mitral stenosis and hypertensive heart disease with similar degrees of congestive failure and compared the results with those of a group of pulmonary emphysema. Their findings in mitral stenosis were similar to other investigators' (12,13, 39). Most of their hypertensives - five out of eight - had audible wheezing respirations and showed ventilatory patterns qualitatively similar to pulmonary emphysema with low M.B.C., air velocity indices, etc. Their results would indicate that cardiac asthma was a very frequent finding in the failure of hypertensive heart disease and they implied that this was true of other diseases of the left ventricle. This was contrary to the finding of others (39,40) and further studies of congestive failure due to valvular and left ventricular disease are definitely needed to clarify the situation.

IV. Blood Gases

The oxygenation of arterial blood expressed as oxygen content,

saturation or as oxygen tension was normal in most cases of cardiac failure (27,35). In severe decompensation there might be undersaturation (41-44) which improved with O_2 therapy (44) or cardiac compensation. The venous O_2 content was often low (42) and the A-V O_2 difference increased in marked congestive failure (43). Anoxemia might be quite marked in cardiac asthma or pulmonary oedema (14,27).

The partial pressure of carbon dioxide in the arterial blood (pa CO_2) provides an accurate measure of the partial pressure of carbon dioxide in the alveolar air, and this in turn varies inversely with the alveolar ventilation. A normal figure of 40 minutes of Hg is exceeded when alveolar hypoventilation exists and is low with alveolar hyperventilation. Arterial pCO_2 was usually reduced in congestive failure (34,35) and returned to normal after treatment of failure (45). Only in cardiac asthma and pulmonary oedema did CO_2 retention occur (14,27).

Fixed acid and lactic acid were said to be high in congestive heart failure (46,47) but improved techniques have shown no abnormality (35,48).

V. Distribution and Diffusion Characteristics

Abnormalities of blood gases in congestive failure might be the result of alterations in the distribution or diffusion characteristics of the lungs of such individuals. An index of pulmonary mixing was derived from measuring the percentage nitrogen in alveolar air after breathing 100% oxygen for seven minutes. This index was normal in mitral stenosis despite the finding of residual volume/total lung capacity ratio of over 40% in some cases. Hyperventilation could mask an increase in the physiological dead-space.

Several workers (49-53) showed an increase in the gradient between the partial pressure of oxygen in the alveolus and in arterial blood.

This could be due to venous admixture - i.e. contamination of anaerated with unaerated blood due to perfusion of non-aerated lung segments; due to dead-space-like ventilation or to some abnormality of the alveolar capillary 'membrane'. Williams (51) found venous admixture to account for the increased gradient, but others found abnormalities in the other parameters (14,50,52).

Carrollet al (50) classified cases of mitral stenosis into four groups. Group I was asymptomatic and had normal distribution and diffusion. Group II had normal diffusion but venous admixture and dead-space-like ventilation indicated distribution defects. These patients had moderate symptoms with slightly decreased vital capacities and maximal breathing capacities. Group III had abnormal diffusion but normal distribution and normal vital capacities, maximum breathing capacities and moderate symptoms. Severe disability marked the fourth group and was accompanied by abnormal diffusion and distribution. Reduced vital capacity was found in all and reduced maximum breathing capacity in half. Maximal diffusing capacity (during exercise) was not measured but the changes found were felt to be outside the range of variation of the resting values. An abnormal gradient was shown by Blount (50) during exercise but venous admixture and diffusion defects were not differentiated. Fowler (52) studied 13 patients with mitral stenosis and moderate disability: five had abnormal diffusing capacity, five showed venous admixture, and six had dead-space-like ventilation. Unlike Carrollet al, they could not correlate their measurements with either pulmonary venous pressure or the clinical state.

The cause of abnormal distribution and diffusion in congestive heart failure might be structural or functional (50). Obstructed bronchi, re-

current pulmonary emboli, pulmonary fibrosis or capillary closure might play a part. Changes in alveolar walls, capillaries, capillary basement membranes, as well as vascular changes, have been described in the lungs in mitral stenosis and left ventricular failure (54-56).

On the other hand, functional changes such as oedema of the air passages, fluid in the alveoli or changes in vascular pressure might play a part. Carroll found some correlation between diminished diffusing capacity and elevations of pulmonary arterial pressure (57), while others (49, 53) could not support such a relationship.

VI. Pulmonary Mechanics

Diminished lung distensibility has been forwarded as the main factor in the diminished vital capacity (9,11,16,39,40,58), in the decreased maximum breathing capacity (13,27,50), and as one of the factors in abnormal distribution and diffusion found in marked congestive failure (50). The study of the stress-strain relationship of the lungs in congestive heart failure might, therefore, be informative.

Carson, in 1820, (59) noted a retractive force of the lungs when the chest was opened in cadavers, but the pressure-volume curves of lungs from dead animals were found to be erratic (60). Over the physiological range of distension, however, pressure was nearly a linear function of volume (61). Roher, in 1925, (62) made the classical descriptions of how mechanical properties may be separated into different components. Elastic forces within the respiratory system depended on the degree of lung deformation and hence on volume change. At the same time he considered forces necessary to overcome the frictional resistance within the system (gas flow and tissue frictions resistance) to be related not to volume but to rate of change of volume. Similarly, the forces concerned with

acceleration of mass would be related to dynamics of the lung. In the static state, with constant lung volume, only elastic forces should appear. Confirmation of this concept in both animals and humans came from several workers (63-67). Christie and Meakins (68) found that greater changes in intrapleural pressure were necessary to produce the same tidal volume in congestive failure as in normals. Intrapleural pressure often became positive during expiration in congestives while it was normally negative throughout the respiratory cycle. Further investigation in humans was delayed, probably due to the potential danger of creating pneumothoraces in already ill patients. Mack et al (69), Heyer et al (70) demonstrated in animal preparations that vascular congestion, produced by infusions into the pulmonary vessels, increased the pressure necessary to inflate the lungs. The demonstration that intrathoracic pressure changes might be reflected in intra-oesophageal pressure led to further studies in humans, employing simultaneous recordings of intra-oesophageal pressure and air flow (71,72).

When air flow was zero, the pressure produced by the retractive force of the lung was active. During air flow the pressure required to overcome resistances to flow and tissue friction was also active. Flow resistances were comprised of resistance to laminar and turbulent flow and these could be separated by applying a mathematical equation approximating the pressure-flow curve. Tissue friction could be assessed by breathing gas mixtures of different densities, which would alter only resistances to flow (39,73).

Brown, Fry and Ebert (39) have analysed pulmonary mechanics in 17 patients with cardiac disease, and elastic resistance was found increased compared to normal. The decrease in compliance was correlated with the reduction in vital capacity (39,40,73).

Resistance to gas flow was increased in some but not all cardiacs, both laminar and turbulent resistance being affected. Those patients with increased flow resistance had several bouts of congestive failure and their disease was classed as severe. No correlation was established between flow resistance and decreased vital capacity. Tissue friction was considered negligible.

Marshall, McIlroy and Christie (36) measured the work of breathing in patients with different degrees of heart failure due to mitral stenosis. Since a pressure-volume or stress-strain relationship contains the elements of work, they calculated the work done against elastic and flow resistance in gram centimetres. The area of the pressure-volume loop was equivalent to non-elastic work and the area of the triangle subtended by the straight line joining the end points of the loop was equivalent to the elastic work. Total work was increased at rest in cardiacs, and there was a disproportionate rise in these same patients on exercise compared to normals. The increase was predominantly due to elastic resistance, attributable to the rigidity of congested lungs. Employing a formula expressing the mechanical factors of breathing (74), one can calculate an optimal rate of breathing to expend the least work and maintain adequate alveolar ventilation. The rate calculated by this formula closely approximated the actual respiratory rate observed in cardiac and normal controls at rest and on exercise. These workers concluded that patients with congestive failure breathed at a rate and depth most economical in terms of respiratory work. Thus the shallow breathing associated with cardiac dyspnoea required less work because of the large increase in elastic resistance. Marshall, Stone and Christie (75), employing the same methods, compared the performance of normals and cardiac patients during exercise which was sufficient to produce enough respiratory

distress to stop the exercise. They found normal subjects could increase respiratory work to a greater extent than cardiacs. This could be reproduced by having normal subjects breathe through a resistance. Measurements of the changes in intra-oesophageal pressure showed that about the same force, in cm. of H_2O, was exerted by cardiacs and normals when both reached the point of maximal dyspnoea. They suggested that a 'maximal force' was a common factor in the dyspnoea of both produced by exertion. Normal subjects, however, can achieve a much higher minute volume before reaching this point. Increased resistances to breathing are, therefore, suggested by this study though they were not analysed. The amount of force which could be applied to the lungs would depend on the muscular and nutritional state, the emotional state as well as the mechanics of the lungs of an individual.

Reduction of the total intrathoracic space by such things as an elevated diaphragm, cardiac enlargement, pleural effusion and ascites would decrease the volume of the lungs but would not alter the elastic properties of the lung. Pressure changes required to overcome the elastic resistance would be expected to be less at lower levels of lung inflation, whereas an increase in pressure requirements is actually observed.

Increased pulmonary blood volume has been suggested as the cause of lung rigidity (17,18,19,69,70). Infusions into the pulmonary vessels of the lungs of animals increased the pressure required to produce the same inflation (69,70). An increase in the vital capacity of the recumbent patient has followed venesection or pooling of blood in the extremities (19-21). Encroachment on the alveoli by engorged blood vessels was proposed as the mechanism by which blood volume affected vital capacity, and it was said that the decrease in vital capacity was equal to the amount

of blood added to the pulmonary circuit (11). Increases of pulmonary blood volume of this order were not found in mitral stenosis (17,18). An alternate explanation may be interstitial oedema and rigidity produced by an increased pulmonary capillary pressure (39). No correlation was found between either pulmonary 'capillary' pressure or pulmonary artery pressure and compliance (40,76). However, infusions of albumin and other procedures designed to change the volume of blood in the lungs have raised the pulmonary vascular pressure and reduced the distensibility (77,78).

Increased resistance to flow seen in some cases of congestive heart failure might be due to oedema of the bronchial walls and to fluid lining the bronchioles (39). Obstruction of bronchioles in some areas of lung would necessitate hyperventilation of remaining portions of lung. This in turn would mean higher rates of flow and greater pressure gradients to produce them. The use of intra-oesophageal pressure to indicate intrathoracic pressure is not completely on solid ground and there is evidence that the absolute intra-oesophageal pressure may be quite different than intrapleural pressure and may be influenced by other than changes in pressure within the thorax (79-81). Studies of mechanics in different etiological types of cardiac disease and classification according to severity of failure would help in our understanding of the mechanics of the lungs in congestive heart failure.

VII. Conclusion

Impairment of respiratory function in congestive heart failure cannot, in the light of our present knowledge, be ascribed to a single factor. The changes in lung volumes and, in particular, the diminished vital capacity (which was once considered the major disability) are not indicative of clinical impairment of function. Studies of pulmonary dynamics are ab-

normal in severe failure but again do not parallel the symptoms. Patients with very similar disability may have quite different diffusion and distribution characteristics. Changes in pulmonary elasticity and resistance to flow may be present in heart failure but have not been studied in sufficient numbers to permit correlation with the symptoms.

Measurements of pulmonary function may be surprisingly normal in patients with considerable dyspnoea but seem to be increasingly impaired due to a variety of causes as the severity of the failure progresses.

It seems clear from this review that further studies are needed to explain the mystery of cardiac dyspnoea.

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Part II. An Experimental Study of the Effect of Posture on the Mechanical Properties of the Lungs in Congestive Heart Failure

I. Introduction

The mechanical properties of the lungs which oppose the action of the respiratory muscles consist of two types of resistance — elastic and viscous. Both these are operative during lung movement but under static conditions only the elastic resistance is reflected in the pressure required to maintain any given state of inflation. During movement the viscous resistance comes into play and its additional effect on the pressure depends on both the resistance to air flow and the resistance to distortion of the non-elastic elements of the lung. Thus, by measuring the pressure differential across the lung under appropriate circumstances it is possible to obtain estimates of both the elastic and viscous properties of the lung.

In 1917 Peabody (1) ascribed the dyspnoea of congestive heart failure to an abnormality of the mechanical properties of the lungs. More recently orthopnoea has been explained on a similar basis, an increase in the elastic resistance being thought to be an important factor (2,3,4,5). The purpose of this paper is to report measurements of the elastic and viscous resistances in the erect and supine position. These show that the viscous resistance may be of greater consequence than the elastic in the genesis of orthopnoea.

II. Method

The subjects were five normal individuals and five patients with congestive heart failure due to rheumatic or arteriosclerotic heart disease (Table I). At the time of study all patients had some degree of respiratory distress while lying down in addition to venous distension, basal pulmonary congestion, hepatomegaly and peripheral oedema. They also had

a reduction in vital capacity and maximal breathing capacity as measured in the sitting position when compared to the predicted values calculated by the methods described by Baldwin, Cournand and Richards (6).

The mechanical properties of the lungs were measured and calculated by methods essentially the same as employed by Cherniack (7). The rate of air flow into and out of the lungs was measured with a pneumotachometer. The volume of air breathed was determined by measuring the area under the pneumotachograph with a planimeter. The effective intrathoracic pressure was measured by means of a strain gauge transducer connected on one side to an air-filled thin-walled latex balloon in the lower third of the oesophagus and on the other side to the mouthpiece of the pneumotachometer. Tracings of the air flow and oesophageal-mouth differential pressure were recorded on a Sanborn polyviso recorder. Simultaneous measurements of the pressure and volume changes during a complete breathing cycle, determined at intervals of 0.2 seconds, were plotted one against the other. This produced a loop from which the following information was derived.

The pressure required to overcome the elastic resistance of the lungs during the breathing cycle was determined from the slope of a line joining the points of no flow at end expiration and end inspiration. The elastic properties of the lung were expressed as the index of elastic resistance, which is the number of centimeters of water pressure change required to produce and maintain a change of one litre in lung volume. The mechanical work required to overcome elastic resistance was obtained from the area of the right-angled triangle whose hypotenuse was formed by the line joining the points of end expiration and end inspiration as described by McIlroy, Marshall and Christie (8), and was expressed in kilogram centimeters.

The pressure required to overcome viscous resistance at any particular time during a respiratory cycle was ascertained from the pressure-volume

loop by subtracting from the intrathoracic pressure the pressure required to overcome the elastic resistance at that time. The pressures developed against the viscous resistance were plotted against simultaneously recorded rates of air flow and the pressure existing at flow rates of 30 L/min. was chosen to express the viscous resistance. The area of the pressure-volume loop represented the mechanical work done on the lungs in order to overcome viscous resistance and was expressed in kilogram centimeters.

The total work done on the lungs was calculated by adding the elastic work, the inspiratory viscous work and the portion of the expiratory viscous work that fell outside the elastic work area. The work per minute was calculated by multiplying the work per breath by the respiratory rate.

The index of elastic resistance was calculated from the average of ten to twenty breaths. Viscous resistance and the work done on the lungs was calculated from the average of four to five breaths.

The subjects were studied in the sitting position and again five to fifteen minutes after the assumption of the supine position. Patient L.W. was an exception in that he was supported at any angle of thirty degrees because of his inability to lie flat.

III. Results

Elastic Resistance. Table II shows that in the sitting position the index of elastic resistance was higher in the patients with congestive heart failure than in the normal subjects. Assumption of the supine position increased its value in all the normals and in four of the five cardiacs, the fifth being unable to lie flat.

Viscous Resistance. It will be seen in Table II that the viscous resistance in the sitting position was higher in the cardiacs than in the normal subjects. Figure 1 shows that this was true at all rates of air

flow. The greater viscous resistance in the cardiac patients is indicated by the greater pressure required to produce a given flow.

On lying down an increase in viscous resistance occurred in both groups of subjects (Table II). In normal subjects the increase was spread uniformly over the respiratory cycle but in patients with congestive heart failure the increase was not uniform, being particularly marked at the beginning of inspiration and at the end of expiration. The relationship of viscous resistance to the respiratory cycle in the supine position is shown in Figure 2 for the same subjects as shown in Figure 1. It will be seen that in the cardiac patient a greater pressure was required to produce a given flow at the beginning of inspiration than was required as inspiration progressed. In expiration, a lower pressure was required at the beginning of expiration than was required as expiration progressed. The data in Table II indicate that this was true for all the cardiac patients and was not apparent in the normal subjects.

Work of Breathing. In the sitting position the work performed on the lungs against both the viscous and elastic resistances was greater for the cardiac than the normal subjects (Table III). Similarly, the total work of breathing was much greater in the cardiacs than in the normals.

The assumption of the supine position did not result in an increase in the elastic work of breathing in either group, as a decrease in minute volume offset the increase in the index of elastic resistance shown in Table II. The main effect of recumbency was to increase the work required to overcome viscous resistance. The average increase was approximately 30% in the normal subjects while in those with congestive failure it was over 50% of the already very high value. Consequently in the supine position the total work of breathing tended to be increased in both groups but

the effect was most pronounced in the cardiac patients. It is of interest that the one patient (L.W.) who could not lie flat, did not have an increase in elastic resistance or elastic work but did have a marked increase in viscous resistance and viscous work.

IV. Discussion

The results of this study confirm the observations of others (9,10, 11) that elastic and non-elastic resistance is elevated in congestive heart failure in the sitting position. This is presumably related to the increased pulmonary blood volume believed to be present in this condition (12,13).

The assumption of the supine position resulted in an increase in the viscous resistance and viscous work of breathing that was more pronounced than the increase in elastic resistance and elastic work of breathing. This was particularly marked in patients with congestive heart failure. It is logical, therefore, to relate orthopnoea to the increase in viscous resistance induced by the assumption of the supine position.

An explanation of the effect of the recumbent position on the viscous resistance of the lungs must include an explanation of the peculiar relationship of this resistance to the state of lung inflation. A similar but less marked effect of lung inflation has been observed in normal subjects while breathing in an expiratory position or during forced expiration and in emphysematous subjects during expiration (14). These observations suggest that the high viscous resistance at low lung volumes is due to narrowing of the air passages in this state. Patients with congestive heart failure in the supine position would be especially likely to show this phenomenon because of congestion and oedema tending to obstruct the air passages. As the lungs expand these passages would be opened, lowering the resistance to air flow.

In assessing the effect of recumbency on respiration, consideration should be given to the fact that the brunt of the increase in viscous resistance fell in late expiration and early inspiration. It is felt that this concentration of the resistance to breathing on one part of the respiratory cycle would have a more disturbing effect than if its equivalent were spread over the whole cycle. If this be true, the values for the work of breathing reported in Table III under-estimate the adverse effect of recumbency. In any case, the values show that the total work of breathing of the supine cardiacs was approximately 25% greater than that of the sitting cardiacs and that this difference was attributable to the difference in the work against viscous resistance.

V. Summary

The assumption of the supine position resulted in a marked increase in viscous resistance in patients with congestive heart failure. The major part of this increased resistance occurred at the end of expiration and beginning of inspiration when lung volumes were low. As a consequence, the work of breathing in patients with congestive heart failure in the supine position was increased by 25 percent. The dyspnoea of recumbency in these patients is tentatively attributed to interference with the patency of the respiratory passages at low levels of lung inflation.

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Table I

Clinical Data of Five Normal Subjects and Five Patients With Congestive Heart Failure

Patient	Sex	Age (yr)	Height (cm)	Cardiac Diagnosis*	Vital Capacity (cc) Pred.	Vital Capacity (cc) Obs.	M.B.C. (L/min) Pred.	M.B.C. (L/min) Obs.
NORMALS								
R.N.	M	33	178		4230	5580	124	150
B.B.	M	16	167		4200	3730	133	96
R.K.	M	24	178		4370	6260	132	154
H.J.	M	27	178		4430	5580	132	123
H.K.	M	21	173		4280	6070	132	194
CARDIAC PATIENTS								
H.D.	M	67	170	A.H.D.	-	-	-	-
L.L.	F	44	168	R.H.D.	2950	2237	90	39
S.K.	M	70	173	R.H.D.	3450	2802	82	73
E.H.	M	77	178	A.H.D.	3530	2395	-	-
L.W.	M	66	168	A.H.D.	3320	1160	75	33

* A.H.D. - Arteriosclerotic heart disease
R.H.D. - Rheumatic heart disease

Table II

The Index of Elastic Resistance and the Viscous Resistance in Five Normal Subjects and Five Patients with Congestive Heart Failure in in the Sitting and Supine Positions

Subject	Index of Elastic Resistance (cm H_2O/L)		Viscous Resistance Press. (cm H_2O) to produce flow 30 L/min.					
	Sitting	Supine	Sitting		Supine*			
			Insp.	Exp.	Insp.		Exp.	
					Low	High	Low	High
NORMALS								
R.N.	5.99	6.76	0.6	1.0	0.4	0.9	1.9	1.8
B.B.	9.34	12.50	1.1	1.4	1.3	1.2	2.0	2.3
R.K.	4.20	5.46	1.2	1.1	1.7	1.7	2.7	3.1
H.J.	7.82	11.10	0.9	0.9	1.3	1.2	1.1	1.2
H.K.	3.52	6.50	0.6	0.8	1.2	1.0	1.2	0.9
Mean	5.40	8.46	0.9	1.0	1.15	1.2	1.8	1.8
CARDIAC PATIENTS								
H.D.	10.86	21.74	1.2	1.4	4.25	1.15	6.8	1.7
L.L.	11.5	14.92	3.2	4.1	5.9	3.2	8.8	5.2
S.K.	7.94	9.62	1.8	2.2	5.1	1.7	9.1	2.0
E.H.	10.75	11.00	1.3	2.2	2.6	0.6	5.5	2.0
L.W.	21.30	15.40	1.5	2.4	2.35	2.15	4.4	1.9
Mean	12.47	15.54	1.8	2.5	4.0	1.8	6.9	2.6

* Viscous resistance in the supine position is shown separately at high and low levels of lung inflation because of the marked effect of lung expansion on the viscous resistance seen in the supine cardiac patients.

Table III

The Work of Breathing of Five Normals and Five Patients with Congestive Heart Failure Measured in the Sitting and Supine Positions

	Work of Breathing (Kg cm/minute)					
	Elastic		Viscous		Total	
Subject	Sitting	Supine	Sitting	Supine	Sitting	Supine
			NORMALS			
R.N.	26.4	27.1	26.9	27.9	39.9	41.1
B.B.	11.0	17.2	25.6	17.2	23.8	25.8
R.K.	39.6	34.9	49.6	92.4	64.4	81.1
H.J.	49.4	34.9	39.2	40.9	69.0	55.4
H.K.	32.2	40.7	18.9	37.5	41.7	59.5
Mean	31.7	31.0	32.0	43.2	47.8	52.5
			CARDIAC PATIENTS			
H.D.	57.8	87.6	101.4	152.0	123.7	186.3
L.L.	58.0	66.1	99.0	132.5	122.4	152.1
S.K.	39.2	40.0	36.3	122.5	62.8	119.6
E.H.	120.1	94.9	140.6	180.2	211.6	211.9
L.W.	40.9	35.6	53.8	72.9	75.9	82.0
Mean	63.2	64.6	86.2	132.0	119.3	150.4

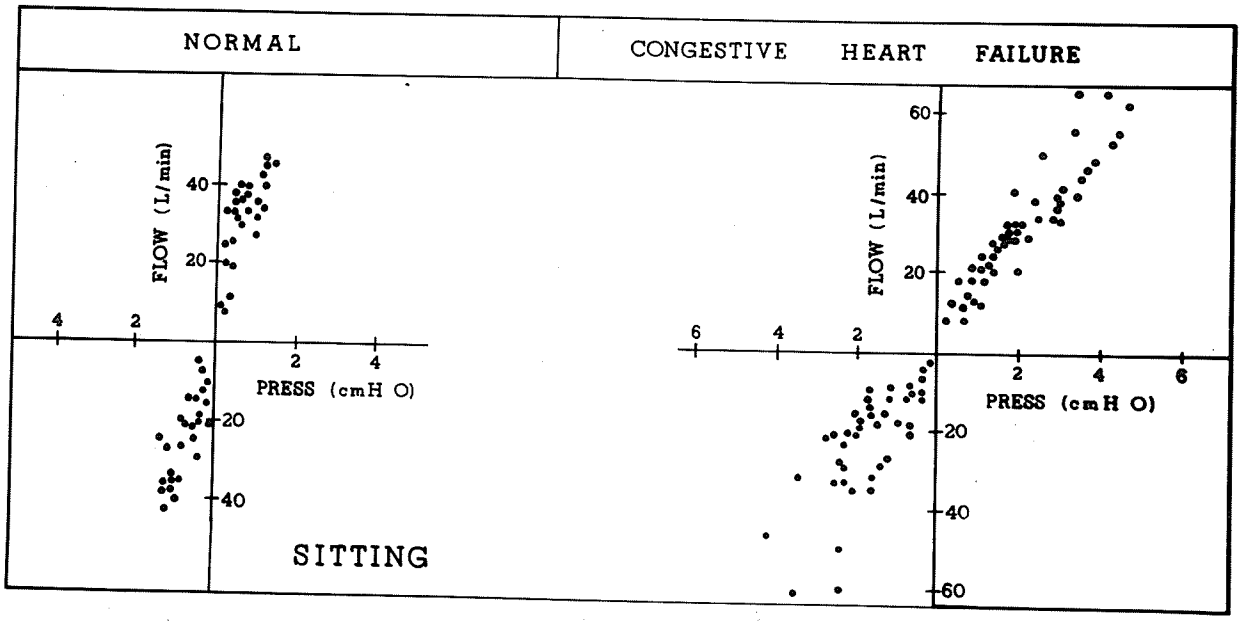

Fig. 1. Viscous pressure-flow curves for a typical normal subject (H.K.) and a patient with congestive heart failure (S.K.) measured in the sitting position. Data from 4-5 breaths.

(Inspiration is represented in the upper right quadrant; expiration is represented in the lower left quadrant.)

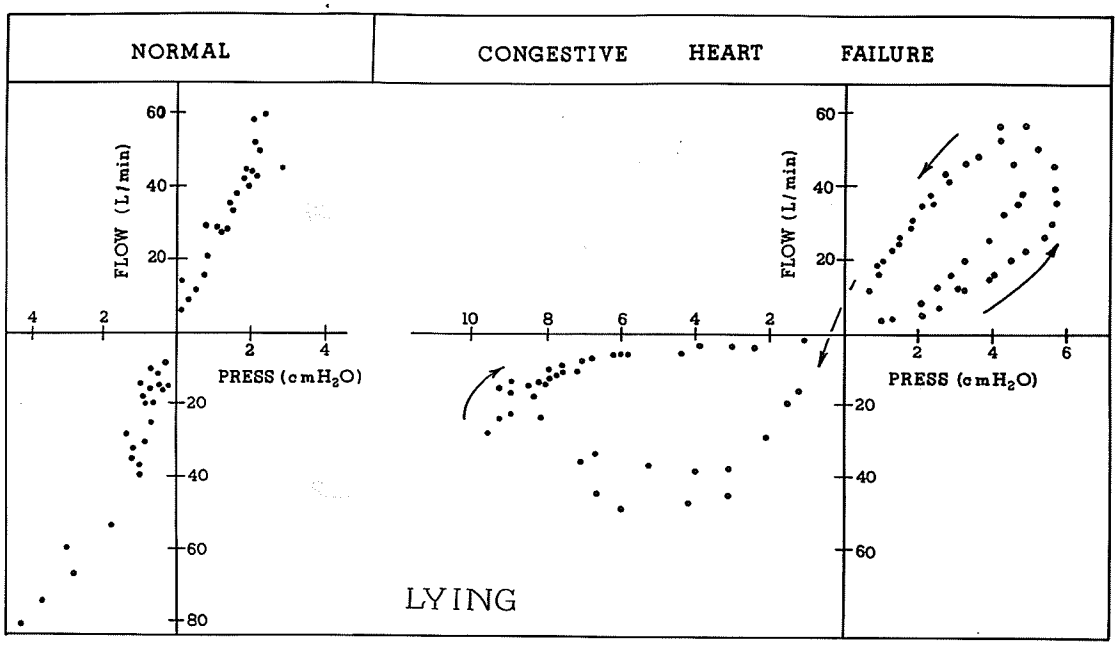

Fig. 2. Viscous pressure-flow curves in the supine position for the same patients shown in Fig. 1. Data from 4-5 breaths.

(Inspiration is represented in the upper right quadrant; expiration in the lower left quadrant.)

Appendix to Part II

Clinical Data on Patients with Congestive Failure

1. Mr. H.D. Age 50. Height 67". Weight 191 lb. Presented with a history of increasing dyspnoea for six months, pain in upper abdomen and lower chest and swollen ankles for six weeks. Abdomen had become larger during this time. No history of severe chest pain.

On physical examination he was a very ill, lethargic, obeseman. His fingers and lips were slightly cyanosed. Neck veins were slightly distended at 30°. His chest revealed bilateral basal moist inspiratory rales. The apex beat was not palpable. The heart sounds were faint. B.P. 140/70. Pulse regular, 100/minute. The liver was palpable 5-6 finger-breadths and was tender. The extremities revealed stasis dermatitis with mild pitting oedema of the ankles and feet.

A chest x-ray revealed cardiac enlargement, heart-lung ratio of 15.8/28.5. Congestion of pulmonary vessels and a small left pleural effusion were present.

The electrocardiogram showed abnormal T waves in leads 1 and 2 and V 5 and 6.

Diagnosis - Congestive heart failure due to arteriosclerotic heart disease.

2. Mrs. L.L. Age 44. Height 66". Weight 174 lb. This woman had rheumatic fever at age 8. She first noticed shortness of breath at age 18. During a pregnancy at age 24 she had episodes of severe dyspnoea, some at night, and had hemoptysis. In the succeeding years she had many hospitalizations for treatment of congestive failure.

Her present admission to the Winnipeg General Hospital was preceded by progressive shortness of breath for six weeks, severe coughing at night and attacks of paroxysmal nocturnal dyspnoea. Had no ankle swelling.

On examination she was mildly orthopnoeic. She had no jugular venous distension. Moist rales were heard in the right lung base. Apex beat was palpable in the 5th interspace outside the mid-clavicular line. She had auricular fibrillation. The apical rate was 104/minute. A grade I systolic and a grade II-III late diastolic rumble without a thrill were heard at the apex. The liver was palpable 2-3 fingers. The chest x-ray showed a heart-lung ratio of 18/30 cm. with some pulmonary vascular congestion. The EKG showed a right bundle branch block.

Diagnosis - Congestive failure due to mitral stenosis and rheumatic heart disease.

3. Mr. S.K. Age 70. Height 68¼". Weight 170 lb. This man had rheumatic fever in 1907 - age 22. He became short of breath in 1934 and had increased dyspnoea with auricular fibrillation in 1937 - age 52. At this time he was digitalized and placed on a salt-free diet. Swelling of the ankles and shortness of breath and cough had prompted his admission to hospital.

On examination he was apprehensive and had dyspnoea on recumbency. His neck veins were just elevated at 30°. Rales were heard at the right base. The apex beat was diffusely thrusting in the 5th interspace, 4½" from the mid-line. A coarse parasystolic murmur and faint early diastolic rumble were heard at the apex. Liver was palpable 2-3 fingers. Extremities showed 1+ oedema. The chest x-ray showed marked cardiac enlargement, mostly left ventricular. The EKG showed left ventricular hypertrophy with T wave changes.

Diagnosis - Congestive failure due to rheumatic heart disease with mitral regurgitation and mitral stenosis.

4. Mr. E.H. Age 77. Height 70". Weight 104 lb. This man had a history of dyspnoea and ankle swelling since a "heart spell" in 1953 - age 75. He had been taking digitalis irregularly and occasional mercurial diuretic. In January 1953 an EKG showed complete A-V dissociation and left ventricular strain. During the month prior to admission this patient had decreasing exercise tolerance and ankle swelling and had paroxysmal nocturnal dyspnoea and orthopnoea prior to admission.

On physical examination he was mildly orthopnoeic. Fundi grade ii. Slight jugular venous pressure elevation with fine rales heard at both lung bases. His B.P. was 195/95. Pulse 64, regular. No pulse deficit. Apex beat outside mid-clavicular line in the 6th interspace. Grade II soft short systolic murmur was heard at the apex. The liver was palpable 2 fingers. Market pitting oedema was present to the mid-thighs.

A chest x-ray showed increased heart size; heart-chest ratio 18.2/30. Left ventricle prominent. The aorta was elongated. Root shadows prominent with increased markings in the right base. The EKG showed complete A-V dissociation with left ventricular hypertrophy and strain plus digitalis effects.

Diagnosis - Congestive heart failure due to arteriosclerotic heart disease.

5. Mr. L.W. Age 65. Height 66". Weight 135 lb. This man had a myocardial infarction in May 1953 and was treated for congestive heart failure by a country doctor following this.

In December 1953 he was admitted to the Winnipeg General Hospital in congestive failure. He was found to be mildly hypertensive (160/90) and

an EKG showed a recent antero-septal infarction plus left ventricular hypertrophy.

Since June 1954 the patient had again been dyspnoeic on exertion. He had ankle oedema since November 1954 and had been orthopnoeic and bed-ridden since the end of November 1955.

On admission on March 3, 1955 he was dyspnoeic at rest with increased jugular venous pressure. Movement was reduced in his left chest. Fine rales were heard at both bases with coarse rales in the left posterior base. Dullness was present in the left posterior lung base. The apex beat was thrusting in the anterior axillary line in the 5th interspace. B.P. 110/90. Pulse 84/minute, regular. Heart sounds were indistinct; no murmurs were heard. The liver edge was palpable and there was moderate pitting oedema of the extremities.

A chest x-ray showed marked cardiac enlargement with congested pulmonary vascular markings and a wedge-shaped infiltration in the left base posteriorly. An EKG showed an old anterior infarction plus left ventricular hypertrophy and strain.

Diagnosis - Congestive heart failure due to arteriosclerotic heart disease. Probable pulmonary infarction.