RESISTANCES TO BREATHING

Part I. A review of the literature.

Part II. An experimental study of the compliance of the total respiratory system and its components in health and obesity.

by

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Abstract

Part I

A review of the current literature on the resistance to breathing indicates that the total respiratory resistance is made up of component resistances offered by the lung and the chest wall. Lung resistance itself has been shown to comprise elastic and non-elastic components - the latter being due to airway and tissue viscous resistance. While present techniques fall short of the ideal, considerable progress in the understanding of lung resistance has been made. Technical difficulties, however, have hindered study of the mechanical properties of the chest wall to a greater extent and although some information has been obtained concerning the elastic properties of the chest wall, little is known regarding non-elastic resistance.

Part II

A modification of a recently described technique for measurement of the compliance of the total respiratory system and its components was used to study normal and obese subjects. Measurements of lung compliance in normal individuals yielded values which agreed closely with those reported previously. Chest wall compliance was approximately equal to lung compliance

in normal subjects. Lung compliance was normal in the obese but chest wall compliance and consequently total compliance were significantly reduced. Obese individuals demonstrated a further reduction in chest wall compliance during recumbency. The decrease in total compliance in obese subjects was proportional to the reduction in vital capacity noted in these individuals. It was concluded that the increase in mechanical work of breathing in obesity is, in part, due to the increased elastic resistance offered by the chest wall in this condition.

PART I. A REVIEW OF THE LITERATURE

A. Introduction

Until recently, the study of the mechanics of breathing has been approached only superficially although it is apparent that knowledge of the energy requirements and the mechanical work involved in breathing, and the factors which determine them, are of great importance to both the physiologist and the clinician.

The mechanical work performed by the respiratory apparatus during breathing is expended in overcoming various resistances. Knowledge concerning the nature and magnitude of these resistances has permitted quantitative estimates of the mechanical work of breathing to be made.

The present thesis is concerned with the development of the current concepts concerning the resistances to respiration in the human subject and the presentation of results of investigations which seek to extend this knowledge.

B. A Mechanical Analogue of the Respiratory System

The respiratory apparatus is analogous to a two plate piston within a barrel (Figure 1), (1). The opposing surfaces of the two plates are attached to a rubber balloon, the interior of which represents the pleural "space". The retractile force of the lungs is represented by the springs attached to the plate on the left, while that of the chest wall is represented by the springs attached to the plate on the right. The total capacity of the container represents the total lung capacity. An air inlet represents the tracheobronchial tree and the piston represents the respiratory musculature.

It can be seen from this model that in order to create air movement, certain resistances must be overcome. These include inertia due to the continuous acceleration and deceleration of air, that caused by the movement of air through narrow and branching tubes, and that due to the friction of the parts sliding over one another. These resistances are encountered only when air is moving and hence they are called "kinetic" resistances. The resistance due to inertia is small and generally considered to be negligible compared to the other kinetic resistances (2).

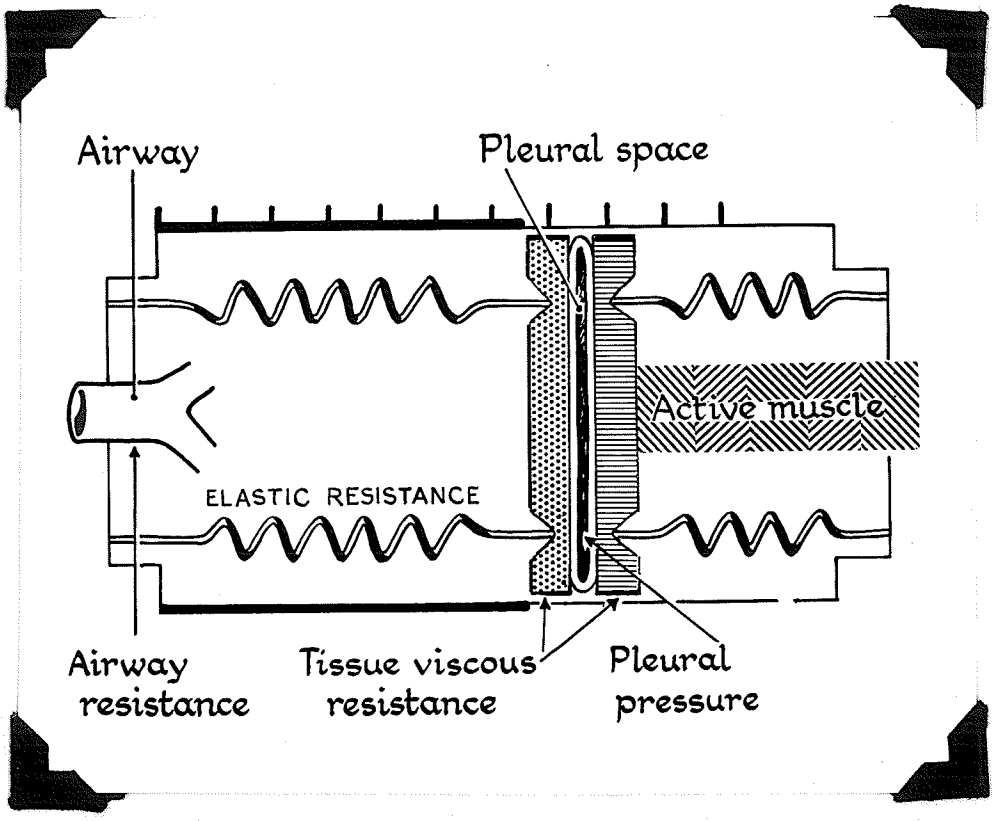

Figure 1. A Mechanical Analogue of the Respiratory System. For explanation see text.

In addition, there are certain forces in operation even when there is no air movement. These static forces are due to the elastic properties of the springs attached to the piston plates. When these forces oppose the movement of the piston they constitute static or so-called elastic resistances. Thus, the total resistance to breathing can be divided into static (elastic) and kinetic (non-elastic) components. The pressure required to overcome this total resistance is given by the formula:

$$P_T = P_S + P_K$$

where P_T is total pressure and P_S and P_K are pressures required to overcome static and kinetic resistances respectively. In order to measure total resistance in such a mechanical model it is necessary to have simultaneous measurements of the differential pressure across the system, the rate of air flow and the volume displacement. While such measurements can be made quite readily using a mechanical model, certain difficulties hinder their determination in the living body.

In order to create air movement in vivo the respiratory muscles contract and exert a force on the lung-thorax system which must be of a magnitude sufficient to overcome static and kinetic resistances similar to those described above. Part of this

force is utilized on the chest wall (thoracic cage, diaphragms, abdominal wall and contents) and part on the lung.

The force exerted on the lung alone equals the pressure applied to the lung multiplied by the surface area. Experimentally this pressure has been estimated by introducing a small amount of air between the parietal and visceral pleura and measuring the pressure in this artificial "intrapleural space" (3, 4, 5). The pressure in such a space varies not only with respiration, however, but is also influenced by the amount of air introduced and the site chosen (5); thus it is only an approximation of the mean pressure applied over the whole lung surface.

Since the measurement of intrapleural pressure is potentially dangerous (8), other methods of estimating transpulmonary pressure in vivo have been employed. Buytendijk (6) reviewed early literature which suggested that the oesophagus reflects intrapleural pressure rather closely and did much of the original work on this subject. Several workers (7 - 14) have since demonstrated that although the relationship between oesophageal and intrapleural pressure is not a constant one and varies from individual to individual, it is close enough so that qualitative studies can be carried out. How closely either of these pressures approximate transpul-

monary pressure is largely unknown; the order of magnitude, however, is generally considered to be the same.

In addition to intrapleural and oesophageal pressure, changes in transpulmonary pressure have also been estimated from changes in venous pressure (15).

The force or pressure applied to the lung-thorax system as a whole is less readily measured than that applied to the lung alone. Whereas normal forces exerted on the lung alone can be made to act through an area in which the resultant pressure can be recorded (intrapleural space or oesophagus), those exerted on the whole lung-thorax system cannot. This is because no method exists by which the force exerted by the respiratory muscles can be integrated. To circumvent this obstacle, methods have been devised in which the normal force of the respiratory muscles is replaced by one of known magnitude from an external source. The resultant pressure applied to the whole lung-thorax system can then be determined. These methods will be described in more detail later.

C. Total Respiratory Resistance

As in the mechanical analogue previously described, the total pressure variation across the lungs and chest wall (P_T) required to inflate the relaxed chest is the instantaneous sum of the pressures required to overcome static (P_S) and kinetic (P_K) resistances, and can be expressed by the formula:

$$P_T = P_S + P_K \qquad (1)$$

Two methods have been employed to study the total resistance to breathing as reflected in P_T. Butler (17) ventilated anesthetized curarized subjects through an endotracheal tube, and found that a pressure variation of 12 cm. H_2O was necessary to achieve ventilation at 20 cycles per minute and a tidal volume of 0.5 liters with a sine wave flow pattern. Otis et al (16) had previously artificially ventilated relaxed, normal subjects in a Drinker respirator. By substituting the data obtained by Otis using the respirator technique in his own calculations, Butler found that the "total hindrance pressure" was less in normals (5.6 cm. H_2O) ventilated in a respirator without anesthesia, muscle relaxants or endotracheal intubation, than in anesthetized subjects. The nature of this difference has not been fully elucidated but on the basis of a later study is probably due to an

increased resistance offered by the lungs during anesthesia (38).

In applying the above estimates of total respiratory resistance to physiological circumstances, one must assume that during ventilation in a respirator, complete relaxation is achieved. It must also be assumed that the pattern of distension of the thorax during artificial ventilation in a respirator or under anesthesia is the same as that which occurs during normal breathing. There is no evidence available which suggests that either of these assumptions is valid.

D. Non-Elastic (Kinetic) Resistance

1. Preliminary Considerations

The pressure required to overcome the total non-elastic resistance of the respiratory system is the instantaneous sum of all the pressures required to overcome the various flow resistive forces encountered during breathing. Theoretically this pressure could be determined by subtracting the elastic pressure from the total respiratory pressure at a given point during the respiratory cycle. The total non-elastic pressure so derived is made up of the pressure required to overcome the non-elastic resistance of the lung and that required to overcome the non-elastic resistance of the chest wall.

Actual measurements of total non-elastic resistance have not been made as yet, partly because of the theoretical and practical limitations of current methods for measuring total respiratory pressure alluded to previously. As a result, information is also lacking concerning the non-elastic resistance of the chest wall (which is derived by subtracting that of the lung from total non-elastic resistance).

The non-elastic resistance of the lung itself, on the other hand, can be obtained in the following manner. From simultaneous

recordings of lung volume and transpulmonary pressure during a respiratory cycle, a pressure-volume loop can be constructed (Figure 2). It can be seen that at any given point during inspiration the total pressure (B_1) is made up of the pressure required to overcome elastic resistance of the lung (B), and that required to overcome non-elastic resistance (B_1 - B). If the latter is now plotted against the rate of flow, a curve is obtained (Figure 3) which represents the non-elastic resistance of the lung. The effect of increasing non-elastic resistance (by narrowing the airway) on the curve is also shown. An alternative method of estimating total non-elastic resistance of the lung consists of momentarily interrupting the flow of air during a respiratory cycle. The resultant change in intrapleural pressure is assumed to equal the non-elastic pressure corresponding to the rate of flow achieved immediately prior to interruption.

The non-elastic resistance of the lung is made up of two components; the resistance offered by the airways and the frictional resistance offered by the lung tissues themselves (tissue viscous resistance).

2. Resistance of the Airway

Investigation of the nature and magnitude of the resistance of the airway has progressed from the theoretical concepts derived

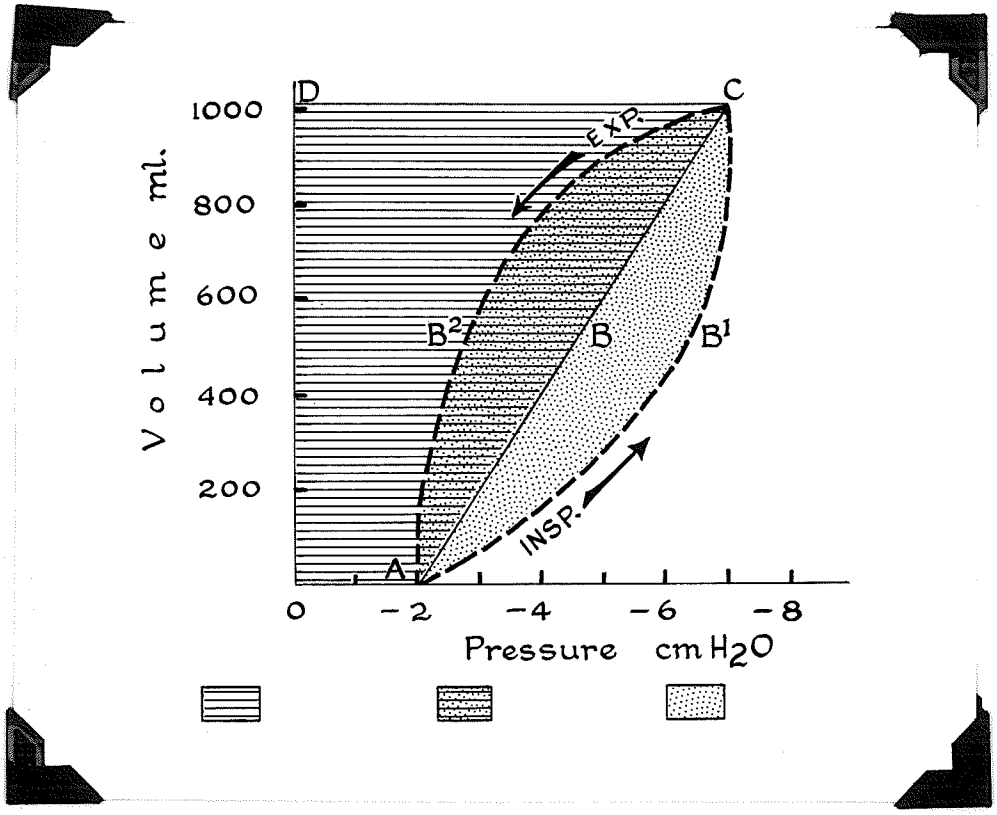

Figure 2. Pressure Volume Loop During a Respiratory Cycle. At any point during inspiration the total pressure (B_1) is made up of elastic pressure (B) and non-elastic pressure ($B_1 - B$).

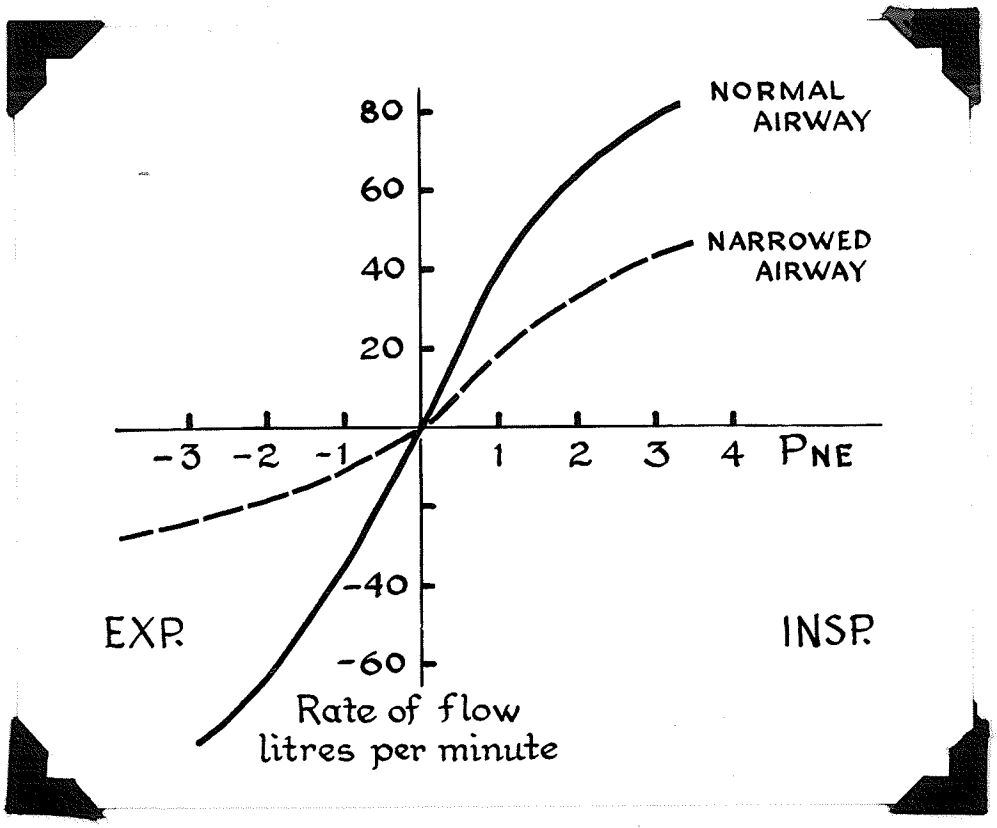

Figure 3. Non-elastic pressure plotted against the rate of airflow with a normal airway and with a narrowed airway.

from a study of the anatomic structure of the tracheo-bronchial tree on excised lungs, to attempts to study airway resistance during "normal" breathing.

The theoretical aspects of airway resistance were first studied in detail by Rohrer (18, 19, 20) who measured the lengths and diameters of the various sections of the tracheo-bronchial tree and calculated the resistance of the whole system. He concluded that nowhere in the airway does flow reach the critical velocity at which it becomes turbulent. Turbulence does in fact occur, however, and Rohrer attributed this mainly to air changing direction rapidly and the successive narrowing of the diameter of the airway. The complete equation he proposed for the pressure drop along the airway due to laminar and turbulent flow was:

$$P = K_1\dot{V} + K_2\dot{V}^2 \qquad (2)$$

The first term represented pressure required to overcome resistance due to laminar (streamline) flow and the second term the pressure required to overcome the resistance due to turbulence. K_1 designated a constant which included both the geometry of the respiratory tract as a system of tubes and the viscosity of the gas, and K_2 included the cross-sectional and bend geometry and also the density of the gas. Since Rohrer's investigations, however, Gaensler (21) has detected an error in Rohrer's calculations.

Because of this, Rohrer had overestimated the critical velocity for turbulence. Correction of this error makes Rohrer's conclusion that all flow is laminar, invalid.

The second method for estimating airway resistance was devised by Bayliss and Robertson (22) who reasoned that airway resistance, but not tissue resistance, would vary with the viscosity of the gases breathed. They ventilated isolated animal lungs with gases of varying density and viscosity. They then constructed a curve relating the changes in pressure and air flow during artificial ventilation with these various gas mixtures. They reasoned that any resistance present when viscosity and density were zero (obtained by extrapolation) must be due to tissue resistance. The difference between this value and that obtained when breathing air represented, therefore, airway resistance. Studies based on a revised principle of using gas mixtures of different density and viscosity have been carried out more recently in man by Fry et al (10), and McIlroy et al (23). The latter authors pointed out that gas combinations which have equal kinematic viscosities (ratio of viscosity to density) should be selected. Despite this precaution the results obtained using this technique have been at variance, perhaps due to failure to control other factors affecting airway resistance.

In order to determine the pressure required to overcome airway resistance, one must know the pressure gradient between the mouth and the alveoli at any given rate of flow. Another technique for measuring this gradient was originally suggested by Veilleumier (24) and has more recently been taken up and elaborated by Otis and Proctor (25). It consists of momentarily interrupting the flow of air at the mouth by means of a solenoid operated shutter. It is assumed that during interruption, pressure at the mouth will come into equilibrium with that in the alveolae. This change in pressure would, therefore, indicate the pressure gradient required to produce the rate of airflow attained immediately prior to the interruption. When this pressure, obtained at various times during the respiratory cycle, is plotted against the corresponding velocity, a curve is obtained, the slope of which is a measure of airway resistance. The equation derived by Otis and Proctor for the pressure required to overcome airway resistance (P_K) at any given velocity ($\dot{V}$) was:

$$P_K = 3.5\,\dot{V} + 1.5\,\dot{V}^2 \qquad (3)$$

Using these constants, Otis, Fenn and Rahn (15), from data obtained on three subjects who were ventilated in a Drinker respirator, estimated that 28.5% of the resistance to breathing was due to air movement.

However, airway resistance, measured by the interruption technique, and total non-elastic resistance, measured by the oesophageal pressure method, were found to be of approximately the same magnitude (28, 30). This suggested that either tissue viscous resistance is a negligible quantity or that the interruption technique measures more than airway resistance alone. In this connection, it has been pointed out (28) that the alveolar pressure after airflow has stopped is not necessarily the same as that before interruption, since there is adequate time for a major change in alveolar pressure to occur during the transition from flow to no flow.

Recently, DuBois, Bothelo and Comroe (29) have devised a method which may estimate alveolar pressure during flow. Using a body plethysmograph, simultaneous recordings of change in thoracic gas volume and change in mouth pressure, were obtained during respiratory efforts made when air flow was interrupted. Since under these circumstances (i.e. when there is no air flow) mouth pressure equals alveolar pressure; change in thoracic gas volume can be calibrated in terms of "alveolar pressure". During airflow, therefore, for any given change in thoracic gas volume, mouth pressure can be measured, alveolar pressure derived and the difference between them used to calculate airway resistance. Normal subjects tested by this method had a mean airway resistance

of 1.5 cm. H_2O per liter per second.

3. Tissue Viscous Resistance

The principle of the measurement of tissue viscous resistance was first set forth by Wirz (9). If the total non-elastic resistance of lung tissue plus airway is known, the tissue viscous resistance may be derived by subtracting the airway resistance from it.

This viscous resistance has been estimated, therefore, by simultaneous measurement of total transpulmonary pressure and "airway" pressure. However, it has already been stated that using the interruption technique, airway resistance would appear to account for all the total non-elastic resistance, tissue viscous resistance being negligible. The objections to this technique and these conclusions have been discussed previously.

The other method used for distinguishing between airway and tissue resistance is by ventilation of the lungs with gases of different viscosities and densities. Using this method, Bayliss and Robertson (22) and others (26) estimated that 75% of the non-elastic resistance to breathing was due to tissue viscous resistance in cats. The high values obtained by Bayliss and Robertson may, in part, be attributable to partial collapse and uneven expansion of the lungs in the open thorax. Furthermore, these studies may be invalid since, as has been noted earlier, the

kinematic viscosity (the ratio of viscosity to density) must be kept constant. This is illustrated by the following investigations.

Fry et al (10) used a mixture containing 80% argon and 20% oxygen having a kinematic viscosity of 0.89 relative to air. They predicted that if there were no tissue resistance, K_1 (laminar flow constant) should increase by 18% over that obtained with air. Their observed value approximated this figure closely, suggesting that tissue viscous resistance was negligible in normal subjects.

McIlroy et al (23), on the other hand, used gas mixtures with a kinematic viscosity of 1.0 relative to air and found a tissue resistance of about 0.6 cm. H_2O per liter per second; i.e. about 30% to 40% of the total non-elastic resistance of the lungs in normal subjects during quiet breathing. The measurement of tissue viscous resistance using foreign gas mixtures, suffers from the disadvantage of requiring measurements of total non-elastic and tissue viscous resistance to be made separately. Furthermore, an effect of the foreign gases themselves on the bronchial diameter, has not been excluded.

Marshall and DuBois (30) have made simultaneous measurements of total non-elastic resistance of the lungs, obtained by intra-oesophageal measurements, and the airway resistance, measured by the plethysmographic technique. The difference between

these two resistances was attributed to the viscous resistance of the lungs. Airway resistance in 12 normal subjects varied from 0.61 to 1.51 cm. H_2O per liter per second; while the total non-elastic resistance varied from 0.82 to 1.68 cm H_2O per liter per second, giving a tissue resistance of 0.10 to 0.35 cm. H_2O per liter per second.

E. Elastic Resistance

From the mechanical analogue previously described (Figure 1), it is apparent that the total elastic resistance to distension has two components; that offered by the retractile force of the lung and that offered by the chest wall. The term chest wall is used to denote all those extrapulmonary structures deformed or displaced during breathing. The elastic properties of the respiratory system are usually expressed in terms of compliance (i.e. the volume change in liters associated with a static pressure change of 1 cm. H_2O), or the reciprocal of compliance called elastance. They may also be graphically represented in a pressure-volume diagram.

1. Total Elastic Resistance

The pressure-volume characteristics of the total respiratory system have been studied in several ways. Among the early attempts were those of Jaquet (32) and Bernoulli (33) who applied rhythmical variations in pressure to the interior of a closed chamber from which the subjects breathed through a tube to a spirometer. By varying the amount of the applied pressure Bernoulli was able to plot the resting pressure-volume diagram of the respiratory system. A modification of this method was more

recently employed by Ferris et al (34, 35), who ventilated subjects in a Drinker respirator in order to obtain pressure-volume curves.

Similar data may be obtained by voluntarily inflating the chest to a known volume, interrupting air flow, and relaxing the chest "against" the interruptor while the pressure at the mouth is measured. The performance of this manoeuvre at various volumes permits the derivation of a pressure-volume diagram usually called a "relaxation pressure curve", (Figure 4). Rahn et al (36) have provided the most complete data on relaxation pressures. Over the normal physiologic range they estimated the slope of the curve to be 0.12 L./cm. H_2O. The curve is shifted to the right on assumption of the supine position, presumably due to the weight of the abdominal viscera. This technique has been criticized because it requires highly trained subjects and, even in such subjects, the results are often variable. Furthermore, it has been demonstrated that complete relaxation is rarely achieved. This latter fact is of great importance since the significance of relaxation pressures depends, to a large extent, on the validity of the assumption that respiratory muscle activity is not influencing the intrapulmonary pressure.

Availability of potent muscle relaxants has enabled workers to study the compliance of the total respiratory system in anesthet-

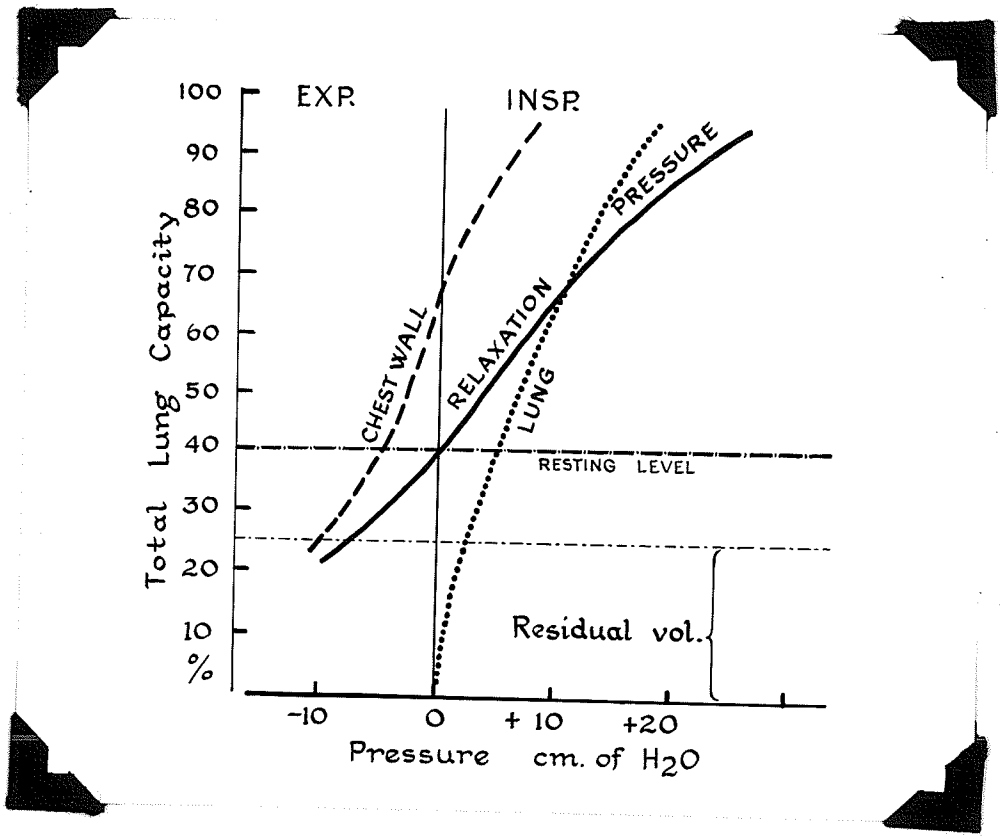

Figure 4. The Relaxation Pressure Curve of the Chest Wall and Lungs.
At the normal relaxation volume or resting level, where the relaxation pressure curve crosses the axis, the elasticity of the lung is exactly balanced by the elasticity of the chest (modified from Rahn et al (35).

ized patients, in the absence of muscular activity. Nims, Conner and Comroe (37) found a decrease in total respiratory compliance in their anesthetized relaxed subjects when compared to unanesthetized spontaneously breathing subjects (0.062 L./cm. H_2O vs. 0.12 L./cm. H_2O). Similar results were obtained by Butler and Smith (38) and others (39). Although this technique solves the problem of adequate relaxation, it is one which employs conditions far from the physiological state.

Heaf and Prime (40), have provided the only estimate of total compliance in the spontaneously breathing unanesthetized normal subject. They applied various continuous positive pressures at the mouth and measured the resultant mean change in end-expiratory volume. In nine subjects the mean compliance of the total respiratory system was 0.09 L./cm. (SD .019). This method does away with the necessity for anesthesia and muscle relaxants, and special training. However, further evidence is required before it can be accepted that the elastic resistance which is determined by this method, is the same as that which is encountered during normal breathing

2. Elastic Resistance of the Lung

The difficulties encountered in attempting to estimate elastic resistance of the whole chest are not encountered when the

elastic resistance of the lung alone is estimated. The pressure-volume curve of the isolated lung was first determined by Lieber-meister (41). Although results were erratic the curve was roughly sigmoid in shape. Similar curves for dog lungs were obtained by Romanoff (42). The sigmoid shape of the curve was more recently confirmed in rats by Lawton and King (43), however, the pressure-volume relationship appears to be a linear one over the range of normal breathing.

The elasticity of the lung in vivo was first studied by Neergaard and Wirz (44) using intrapleural pressure. They pointed out that the pressure required to overcome elastic resistance of the lung could be estimated from interpolated points on the intra-pleural pressure curve at which the velocity of airflow was zero; i.e. where flow resistive forces are not acting.

Two standard methods of measuring pulmonary compliance based on this principle have been used. The so-called "static method" is one in which the transpulmonary pressure (intrapleural or intra-oesophageal) is obtained during interruption of air flow at a given level of lung inflation (usually 0.5 or 1.0 liters) and at the end-expiratory level. "Static compliance" is expressed as L./cm. H_2O pressure difference. "Dynamic compliance" on the other hand, is obtained by dividing the tidal volume of a given respiratory cycle

by the difference in the transpulmonary pressure at instants of zero airflow (i.e. end-inspiration and end-expiration). Lung compliance has been studied by many workers using various indices of transpulmonary pressure including intrapleural and oesophageal pressures.

Measurements of lung compliance depend on whether measurements are made 1) from the normal resting volume, 2) after a deep inspiration, or 3) with the functional residual capacity decreased, either voluntarily or involuntarily, from the effects of posture or anesthesia. Furthermore, Marshall (45) has shown also that compliance in normal individuals is directly related to functional residual capacity, residual volume, inspiratory capacity, vital capacity, body surface area and height. For these reasons, comparison of results obtained by various investigators is difficult. Results obtained by various workers indicate that the mean compliance varies between 0.13 and 0.22 in healthy young adults and is considerably lower in infancy.

It has been demonstrated in isolated animal lungs in living dogs, in normal human lungs after death and in normal living humans, that transpulmonary pressures are less during deflation than inflation (46 - 52). This adaptation of the lungs has been termed

"hysteresis". The term "hysteresis-like" is preferred by Butler (47) because he found that although pressures were greater on inflation than on deflation, no residual deformation was present when the original airway pressure was regained. The hysteresis effect on the lungs has usually been explained, without adequate evidence, on the basis of the overcoming of surface tension forces and the opening and closing of alveolae during inflation and deflation.

3. The Elastic Resistance of the Chest Wall

Unlike the considerable attention which has been directed to the study of the mechanical properties of the lung, those of the chest wall have largely been neglected. The principle of estimating chest wall compliance is a relatively simple one; namely, that if the compliance of the total respiratory system, as well as that of the lungs is known, the compliance of the chest wall can be derived from the difference between them. Difficulty arises, however, in the measurement of total compliance.

The techniques of estimating total compliance by means of relaxation pressures, ventilation of voluntarily relaxed, conscious subjects, or anesthetized, curarized subjects, have already been discussed and the disadvantages and assumptions inherent in these methods noted. The values for mean chest wall compliance obtained by various investigators lie between 0.186 and 0.268

L./cm. H_2O. Further discussion of the compliance of the chest wall appears in Part II of this thesis

The pressure-volume curve of the chest wall is interesting in that frequently smaller increases of tension result from equal changes in volume as the chest is expanded. This has been observed both in anesthetized (38) and conscious subjects (40), but no adequate explanation of this phenomenon has yet been advanced.

The chest wall, like the lung, exhibits hysteresis (47). Unlike the lung, however, there is some residual deformation of the chest wall at the end of deflation when the original airway pressure is regained, and it is said, therefore, to exhibit "true hysteresis".

F. Summary

The resistance encountered during breathing may be described in terms of the pressure differences applied to the respiratory system and the associated volume changes and velocities. The measurement of pressures has proved to be a major obstacle in the study of respiratory mechanics. In studies of total respiratory resistance, inclusion of the respiratory musculature introduces a severe limitation because no practical method exists for integrating the forces of these active muscles. External pumping devices have made exact measurement of the imposed pressures possible only insofar as respiratory muscular activity has been eliminated, either voluntarily or by means of drugs.

In studies of lung resistance alone, the measurement of intrapleural pressures also poses theoretical and practical limitations which are only partly overcome by the use of oesophageal pressures. Similarly, the measurement of airway resistance is hampered by the lack of a practical, direct method for measuring alveolar pressure during airflow. Using such methods as are available it has been possible to delineate the elastic and non-elastic properties of the lung.

Less information has been accumulated concerning the elastic and non-elastic properties of the chest wall. In Part II of

this thesis, a method for studying the elastic properties of the total respiratory system and its components, is described and the results obtained in normal and obese subjects are tabulated and discussed.

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PART II. THE COMPLIANCE OF THE TOTAL RESPIRATORY SYSTEM AND ITS COMPONENTS IN HEALTH AND OBESITY

A. Introduction

It has been noted that marked obesity may be associated with hypoxia and hypercapnia, in the absence of demonstrable intrinsic pulmonary disease, (1-4) and that this is associated with an increased oxygen cost of breathing (5). The latter may be due to either an increased mechanical work being performed on the respiratory apparatus, or a reduced efficiency of the respiratory muscles. The lack of abnormalities in the physical properties of the lungs in obese individuals has led those inclined to the former view to postulate that any increase in total work done is due to an increased resistance offered by the chest wall (5,6,7). The elastic properties of the chest wall in obesity have not yet been studied.

The purpose of this paper is to report studies on the elastic properties of the total respiratory system and its components in a group of obese subjects and to compare them to those of a group of normal individuals.

B. Methods

Thirty-six subjects were studied (Tables I and II). Twenty-four of these were normal individuals, whose mean age was 30 years, mean weight 76 Kg., mean height 176 cm., and mean body surface area 1.94 M^2. Twelve obese individuals, whose mean age was 47 years, mean weight 116 Kg., mean height 170 cm., and mean surface area 2.13 M^2., were also studied. All obese subjects were more than 25% heavier than their ideal weight. None gave a history of pulmonary, cardiac, or neuromuscular disease, and they were asymptomatic aside from somnolence and exertional dyspnea, which were present in a few.

Ventilatory function was assessed in 35 subjects. Vital capacity, inspiratory capacity, maximum expiratory mid-flow rates, and maximum breathing capacity were measured using a Collins respirometer, with the valves and CO_2 absorber removed, and a high speed rotating drum incorporated. The maximum of at least three trials was recorded. Predicted values were determined from the data of Baldwin, Cournand and Richards (3).

The intrapulmonary mixing of inspired gas was assessed by determining the breath to breath washout of nitrogen from the lungs while breathing oxygen, and by measuring the concentration

Table I

PHYSICAL CHARACTERISTICS AND PULMONARY FUNCTION OF NORMAL SUBJECTS

Subj.	Sex	Age (yr.)	Height (cm.)	Weight (Kg.)	B.S.A. (M^2)	Vital Capacity (ml.)	Vital Capacity (%Pred.)	Maximum Breathing Capacity (l/min.)	Maximum Breathing Capacity (%Pred.)	Alveolar N_2 after 7 min. O_2 (%)	Total Lung Capacity (Ml.)
1.	M	24	184	85	2.08	5362	138	198	143	0.7	6260
2.	M	25	180	73	1.98	4950	113	139	105	0.8	5284
										0.8	4800

Table II

PHYSICAL CHARACTERISTICS AND PULMONARY FUNCTION OF OBESE SUBJECTS

Subj.	Sex	Age (yr.)	Height (cm.)	Weight (Kg.)	B.S.A. (M^2)	Vital Capacity (ml.)	Vital Capacity (%Pred.)	Maximum Breathing (l/min.)	Maximum Breathing (%Pred.)	Alveolar N_2 after 7 min. O_2 (%)	Total Lung Capacity (ml.)	Arterial Tension (mm. Hg) O_2	Arterial Tension (mm. Hg) CO_2
A.	F	49	155	104	2.00	1880	76	24	31	0.8	2790	80	57
B.	F	50	155	88	1.87	2260	91	73	95	1.2	3350	76	35
C.	M	57	162	100	2.03	2500	98	62	80	0.6	4310	81	38
D.	F	48	156	103	2.00	2650	105	71	91	0.4	5015		
E.	F	17	165	95	2.01	4115	123	48	48	0.6	4840	67	49
F.	M	34	173	167	2.65	2970	74	55	47	0.7	5030	75	53
G.	F	32	163	91	1.96	3480	113	54	58	0.4	5250		
H.	M	60	161	105	2.06	4005	117	110	120	1.2	4120	81	40
J.	M	70	178	130	2.43	4070	112	111	126	0.4	5190		
K.	F	58	170	116	2.24	2386	86	49	58	0.9	4130		
L.	F	53	165	89.5	1.97					1.8	5385	62	54
M.	M	64	179	93	2.12	3400	91	61	54	1.1	5865		

of nitrogen remaining in the alveolar gas after seven minutes by means of an instantaneously recording nitrogen meter (9). The functional residual capacity (FRC) was determined by measuring the volume and nitrogen concentration of the expired gas at the end of the seven minute period. Total lung capacity was derived by adding the inspiratory capacity to the FRC. The results of the pulmonary function studies are shown in Tables I and II. Fairly marked reduction in vital capacity and maximum breathing capacity was present in some of the obese subjects, while maximum mid-flow rates and nitrogen clearance fell within the accepted limits of normal.

Arterial blood gas analysis was performed in seven of the 12 obese subjects, by the method of Riley et al (10), as modified by Brinkman et al (11). In all, the oxygen tension was low at rest and in two, this was associated with hypercapnia.

In four of the 12 obese subjects the oxygen cost of breathing was determined and found to be elevated (2.5 - 6.2 cc./L). These patients formed part of a series reported elsewhere (5).

The method used to study the elastic properties of the lung and chest wall is a modification of that employed by Heaf and Prime (12). All subjects reported to the laboratory, without special preparation, and were asked to remove any restrictive

garments. A latex balloon was positioned in the oesophagus, and a nose clip fitted tightly. The subject then entered an air-tight body plethysmograph and breathed through a tube into and out of an oxygen filled spirometer, containing a CO_2 absorber, situated outside the plethysmograph.

The oesophageal-mouth pressure differential was used as an index of transpulmonary pressure. A pneumotachometer was incorporated into the airway to record air flow. Recordings of plethysmograph pressure, transpulmonary pressure and airflow were obtained by means of strain gauges leading to three channels of a Sanborn Polyviso recorder, while simultaneous changes in lung volume were obtained from the spirometer tracing.

Following a period of tidal breathing at the resting level, from eight to 20 different continuous pressures, ranging between plus 20 and minus 25 cm. H_2O, were applied around the body by means of a motor blower attached to the plethysmograph. The resultant changes in lung volume and in end-expiratory transpulmonary pressure were noted for each pressure applied, after the breathing became steady at the new lung volume. The mean of five to 10 breaths was used; the end-expiratory point having been determined by the point of zero flow on the pneumotachogram. The various pressures were applied in random order, and the

subject was allowed to return to the resting lung volume after each application of pressure.

In 18 of the 24 normal subjects, and in eight of the 12 obese subjects, studies were performed both in the seated and supine positions. Only negative pressures were applied in 16 normal subjects and eight obese subjects.

An example of the type of records obtained, and the method of their analyses, is shown in Figure 5. It can be seen that a change in plethysmograph pressure of minus 15 cm. H_2O resulted in an increase in lung volume of 1500 ml. and a simultaneous change in transpulmonary pressure of 8 cm. H_2O. The change in pressure across the chest wall was therefore 7 cm. H_2O. Since compliance represents the volume change (in liters) associated with a static pressure change of 1 cm. H_2O, the compliance of the total respiratory system was .100 l./cm. H_2O; the compliance of the lung was .187 l./cm. H_2O and that of the chest wall was .214 l./cm. H_2O.

For each subject lung volumes were plotted against the simultaneous static pressures across the total respiratory system, the lung and the chest wall separately, in the sitting and supine positions. Static transpulmonary pressure at the resting FRC was taken to be zero. A typical example of the pressure-volume

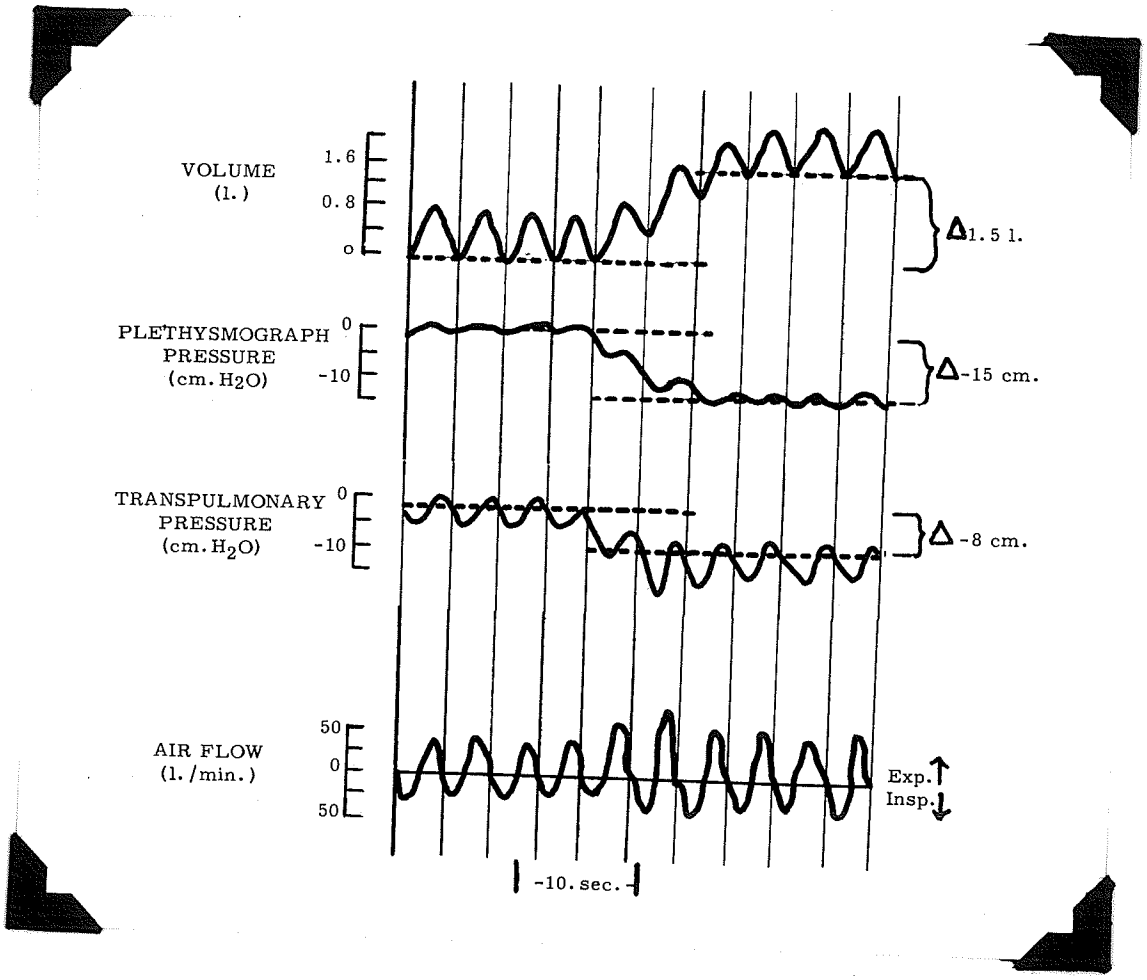

Figure 5. Simultaneous recordings of airflow, transpulmonary pressure, plethysmograph pressure and volume obtained while breathing at ambient pressure and during application of a negative pressure around the body in a plethysmograph.

curves obtained in normal subjects is shown in Figure 6. In addition, compliance was calculated from the part of the curve representing an increase in lung volume of 10% of the total lung capacity above the resting lung volume. In the supine position, compliance was calculated by determining the pressure required to increase lung volume by 10% of the total lung capacity, starting from a lung volume equal to the resting level in the seated position.

Four normal and two obese subjects were each studied on two separate occasions. Compliance values for the total respiratory system, lung and chest wall, determined on the second occasion did not differ from the initial study by more than 15%. No consistent direction of change was noted. The reproducibility of the measurement of volume change and the pressure across the chest wall on the two occasions is shown in Figure 7.

It was assumed during the experiments that the subjects were "relaxed", in the sense that they were not resisting distension of the thorax when pressure was applied. In order to validate this assumption, electromyographic studies were performed in three normal subjects. Suction cup electrodes were positioned over the pectoralis (supramammary), intercostal (Inframammary), scalene, and rectus abdominis (subcostal) muscles, and electromyograms recorded with an indifferent electrode on the arm. After suitable

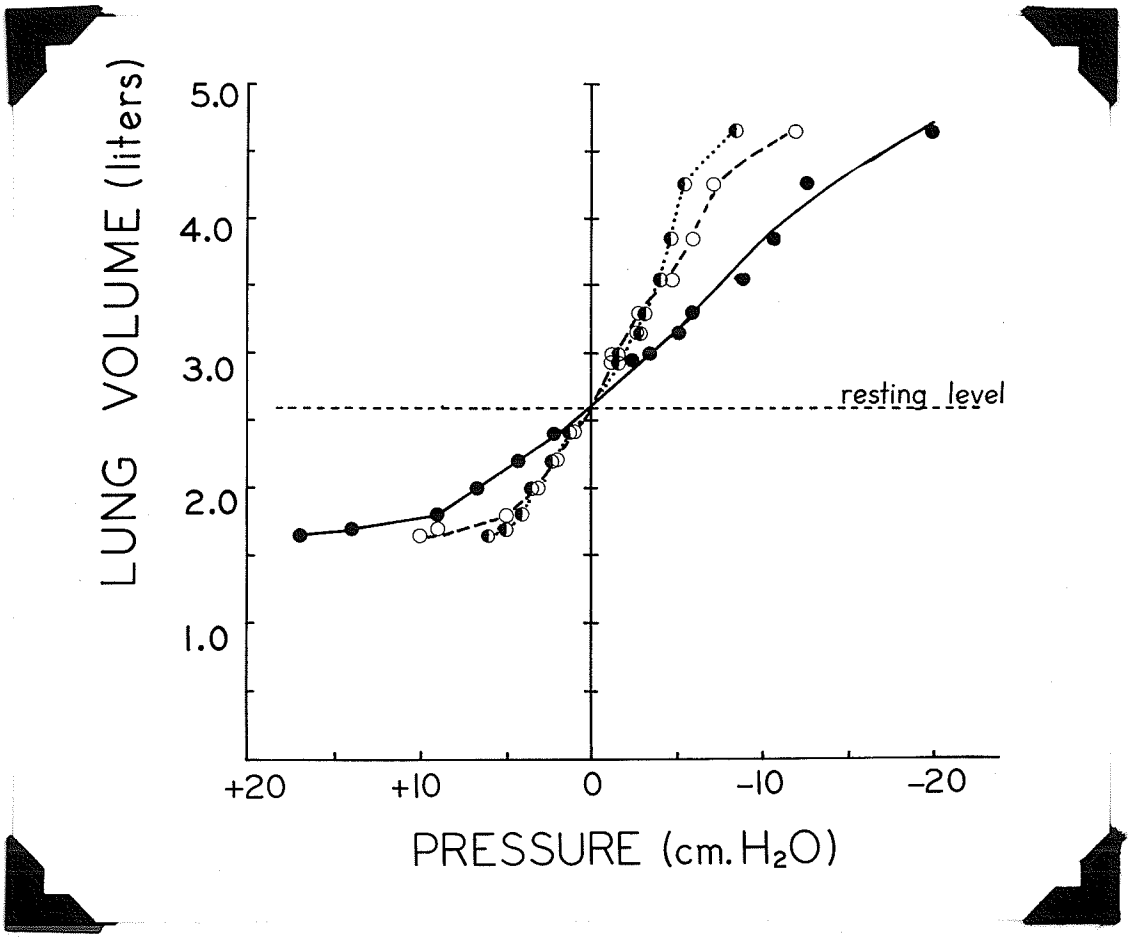

Figure 6. Pressure-volume relationships of the total respiratory system (closed circles), the lung (open circles) and chest wall (half open circles) in a normal subject (14). Plethysmograph pressure represented the pressure across the total respiratory system; oesophageal-mouth pressure, the pressure across the lung and the difference between plethysmograph pressure and the oesophageal-mouth pressure, the pressure across the chest wall.

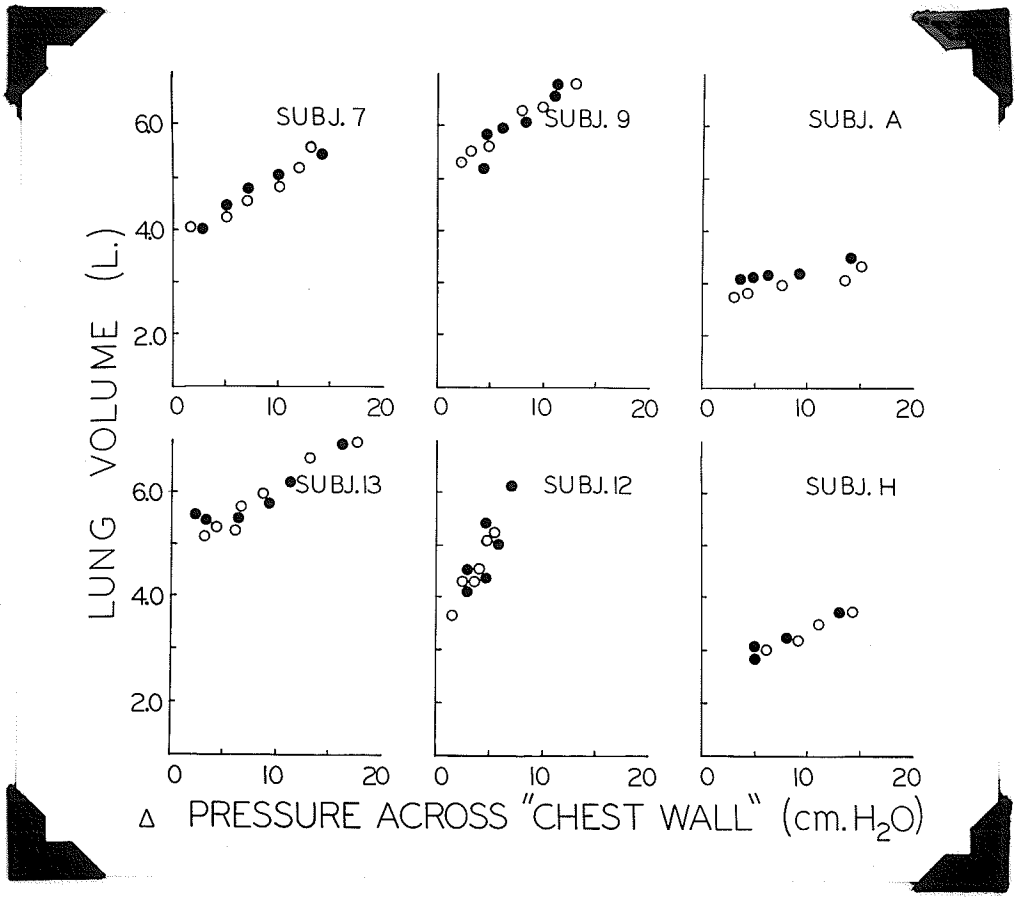

Figure 7. The lung volume plotted against the change in pressure across the chest wall on two separate occasions (closed circles represent the first occasion, open circles the second) in four normal subjects (7, 9, 12, 13) and two obese subjects (A, H).

amplification, these were connected to a Sanborn recorder, on which a simultaneous record of airflow was obtained by means of a pneumotachometer and strain gauge. Records were taken during normal breathing, deep inspirations, while performing "active" (i.e. forced) expirations, and during applications of pressure in the plethysmograph.

The records obtained from the various sites showed similar patterns. Small bursts of activity were occasionally noted during normal inspiration, and these increased during deep inspiration. Normal expiration was free of detectable activity, whereas large bursts of activity occurred during forced expiration. Records obtained during application of pressure in the plethysmograph were indistinguishable from those obtained during normal breathing. No activity was apparent at end-expiration during any of the manoeuvres.

C. Results

Normal Subjects

The pressure-volume curves of the total respiratory system of the lung and of the chest wall for the normal subjects are shown in Figure 8. Since the "slopes" of the curves are an expression of compliance, it is evident that the compliance of the lung and chest wall were nearly equal. It can be seen that the curves for the total respiratory system, the lung and the chest wall were roughly sigmoid in shape. It can also be noted that in some of the normal subjects (e.g. 2 and 5) the pressure-volume curve of the chest wall had an increasing slope over the range of volume achieved.

The calculated compliance values obtained in normal subjects, in both the seated and supine positions, are shown in Table III. In the seated position the mean compliance of the total respiratory system was .119 (S.D. .045) l./cm. H_2O; the compliance of the lung was .283 (S.D. .088) l./cm. H_2O, and that of the chest wall was .224 (S.D. .110) l./cm. H_2O.

Obese Subjects

The pressure-volume curves derived for the obese subjects are shown in Figure 9. It is apparent that the pressure-

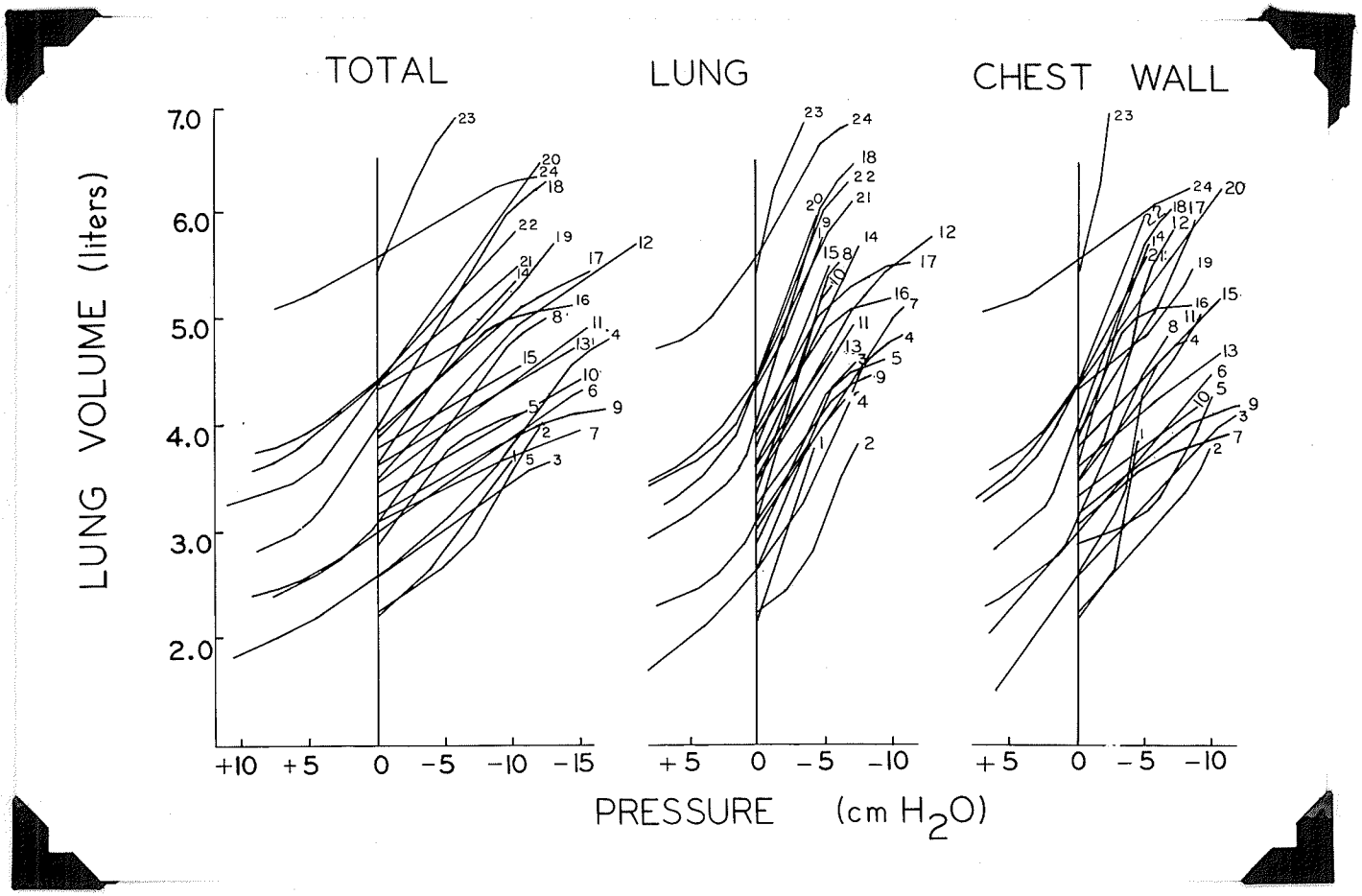

Figure 8. The pressure-volume relationships of the total respiratory system, the lung and the chest wall, in a group of normal subjects.

Table III

RESPIRATORY COMPLIANCE IN NORMAL SUBJECTS

Subject	TOTAL (l/cm. H_2O) Seated	TOTAL (l/cm. H_2O) Supine	LUNG (l/cm. H_2O) Seated	LUNG (l/cm. H_2O) Supine	CHEST WALL (l/cm. H_2O) Seated	CHEST WALL (l/cm. H_2O) Supine
1.	.099	.156	.216	.375	.184	.268
2.	.143	.	.393		.224	
3.	.087	.089	.267	.216	.130	.152
4.	.104		.250		.284	
5.	.072	.050	.154	.107	.135	.268
6.	.075	.088	.369	.316	.094	.121
7.	.085	.130	.198	.329	.150	.214
8.	.067		.140		.104	
9.	.168	.143	.395	.397	.291	.222
10.	.065	.105	.175	.165	.104	.259
11.	.141	.186	.368	.442	.229	.320
12.	.093	.113	.210	.156	.167	.400
13.	.118	.155	.217	.234	.260	.465
14.	.167		.285		.402	
15.	.083		.265		.121	
16.	.127	.120	.196	.168	.360	.430
17.	.215	.141	.441	.234	.346	.370
18.	.106	.104	.225	.240	.198	.184
19.	.123	.123	.425	.309	.173	.224
20.	.173	.140	.362	.293	.333	.269
21.	.107	.143	.268	.291	.179	.274
22.	.138	.097	.323	.293	.242	.149
23.	.228		.390		.550	
24.	.074	.104	.252	.173	.104	.261
Mean	.119	.122	.283	.263	.224	.260
S. D.	.045	.029	.088	.089	.110	.102

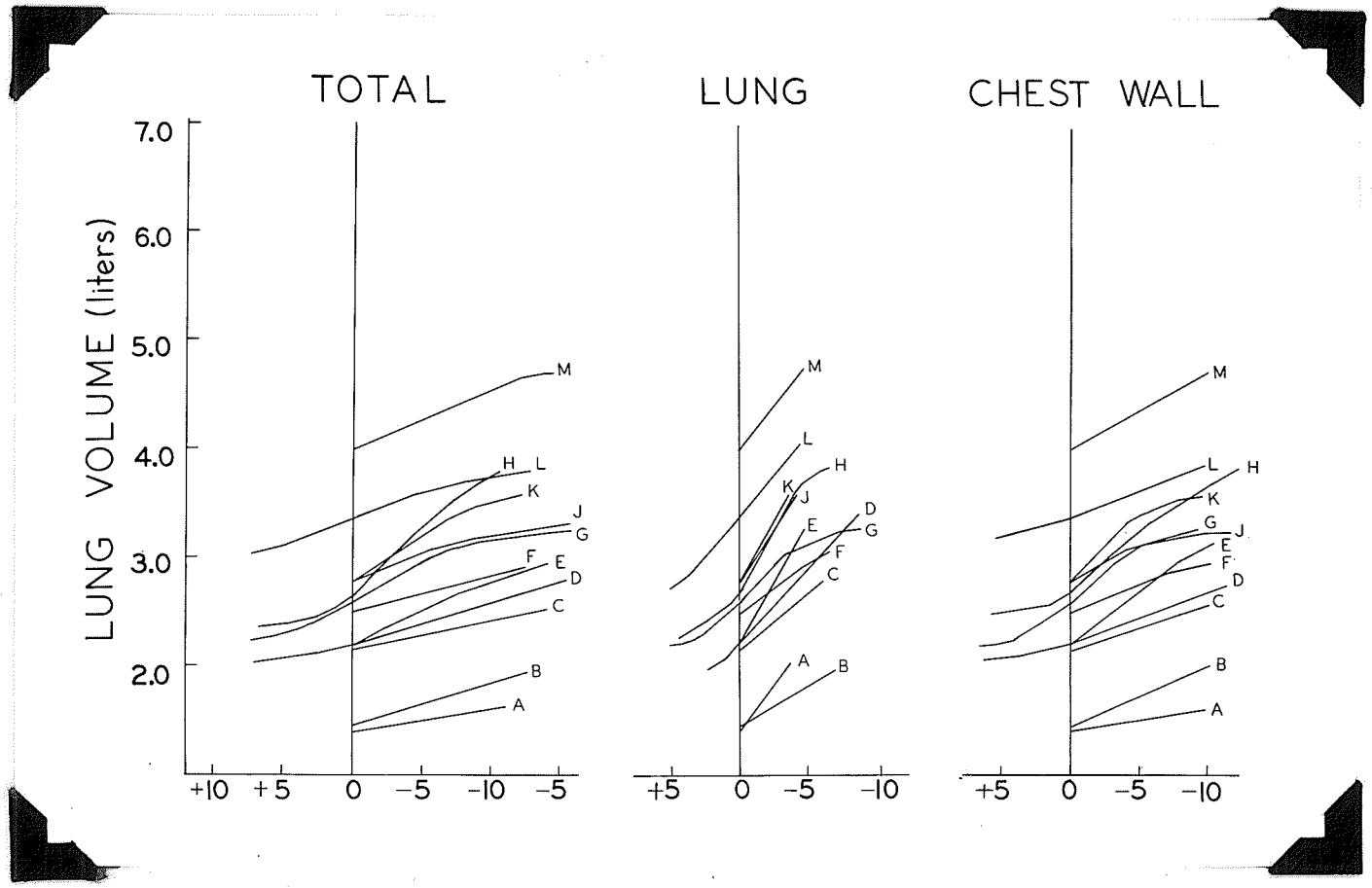

Figure 9. The pressure-volume relationships of the total respiratory system, the lung and the chest wall, in a group of obese subjects.

volume curves for the total respiratory system were flatter in the obese than in the normal individuals. This appears to have been due to a decreased slope of the pressure-volume curves of the chest wall, the curves for the lung being similar to those obtained in the normal subjects.

The calculated compliance values obtained in the obese subjects are shown in Table IV. In the seated position the mean compliance of the total respiratory system was .052 (S.D. .025) l./cm. H_2O; the compliance of the lung was .200 (S.D. .113) l./cm. H_2O, and that of the chest wall was .077 (S.D. .041) l./cm. H_2O. While the compliance of the lung did not differ from the normals, the compliance of the total respiratory system and the chest wall were significantly lower than the normal (p .01) in the seated position.

Since it has been shown that lung compliance is related to vital capacity and FRC (13), it was of interest to correlate the various compliances with these subdivisions of lung volume and with the degree of obesity. This is shown in Table V. It can be seen that the best correlation was between vital capacity and total compliance. This relationship is shown in Figure 10. It can also be seen that the points for normal and obese individuals intermingled and that the relationship between vital capacity and total compliance

Table IV

RESPIRATORY COMPLIANCE IN OBESE SUBJECTS

Subject	TOTAL (l/cm. H_2O)		LUNG (l/cm. H_2O)		CHEST WALL (l/cm. H_2O)	
	Seated	Supine	Seated	Supine	Seated	Supine
A.	.021		.179		.024	
B.	.028		.084		.042	
C.	.025	.027	.119	.090	.031	.044
D.	.059	.052	.193	.129	.080	.068
E.	.047	.045	.138	.123	.071	.071
F.	.059	.035	.140	.136	.103	.047
G.	.077	.057	.175	.164	.138	.085
H.	.077	.056	.246	.246	.111	.072
J.	.107	.037	.367	.412	.151	.042
K.	.034		.103		.052	
L.	.038	.031	.148	.151	.051	.039
M.	.046		.137		.075	
Mean	.052	.043	.200	.181	.077	.059
S. D.	.025	.011	.113	.098	.041	.014

Table V

CORRELATION BETWEEN COMPLIANCE AND VITAL CAPACITY, FRC AND PERCENT OF IDEAL WEIGHT IN NORMAL AND OBESE SUBJECTS

	Compliance					
	Total		Lung		Chest Wall	
	r*	p**	r	p	r	p
Vital Capacity	+.80	.001	+.72	.001	+.74	.001
FRC	+.15	.38	+.44	.02	+.35	.05
Percent of Ideal Weight ***	-.60	.001	-.53	.002	-.55	.001

* r = correlation coefficient.

**p = level of significance of r.

*** Ideal weight obtained from tables published by the Metropolitan Life Insurance Company.

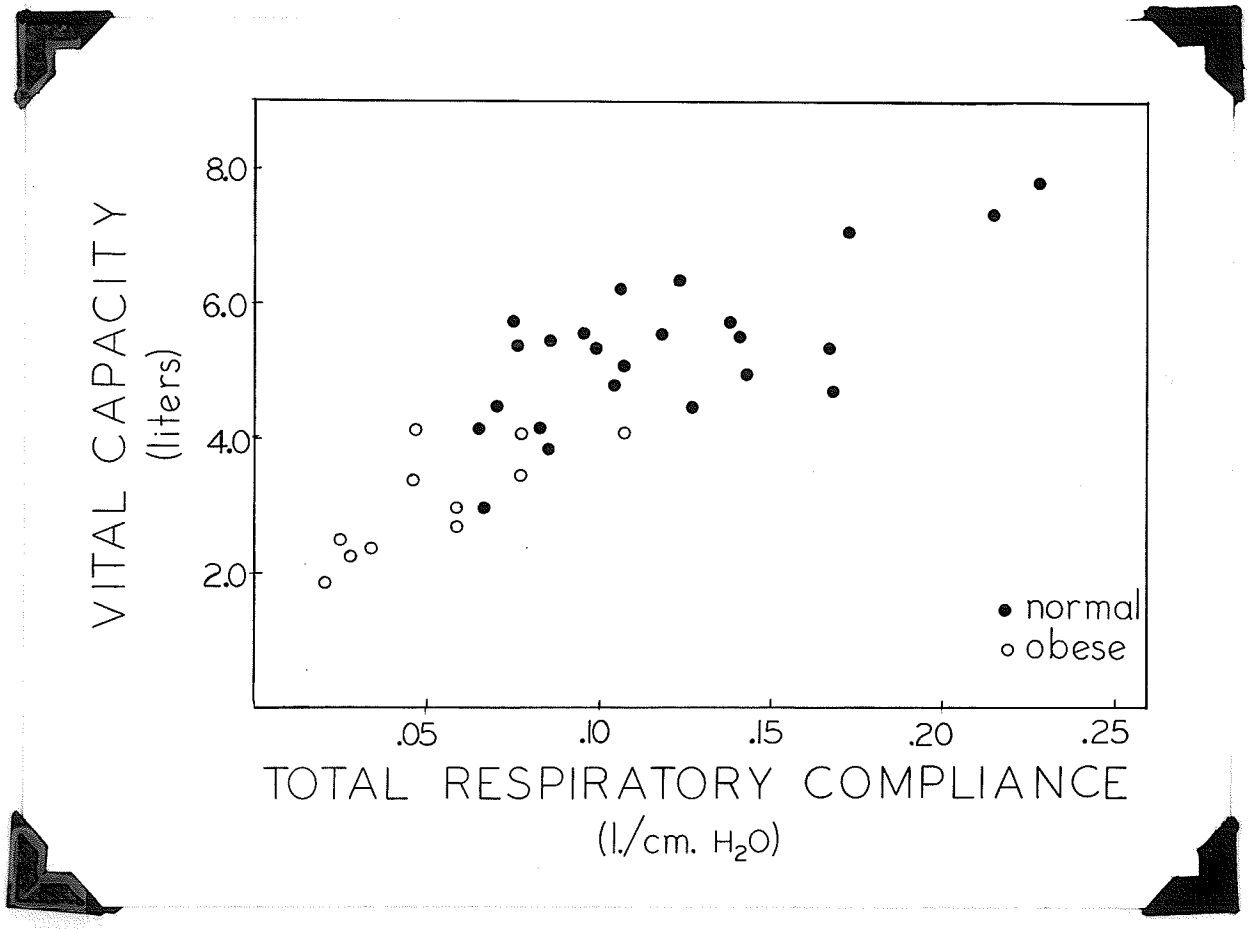

Figure 10. The relationship between vital capacity and the compliance of the total respiratory system in a group of normal and obese subjects.

held true, despite a great variation in the other physical characteristics of the subjects. No correlation could be demonstrated between total compliance and age, height, gross weight or body surface area.

The Effect of Posture on Compliance

The functional residual capacity was reduced in the supine position to a similar extent in both the normal and obese subjects. The effect of posture on the derived compliance values in the normal and obese subjects is shown in Tables III and IV. It can be seen that in the normal subjects the compliance values in the supine position were no different than those obtained in the seated position. On the other hand, in seven of the eight obese subjects studied, the compliance of the total respiratory system and the chest wall was decreased in the supine position. The reduction in compliance of the chest wall in obese subjects on assumption of the supine position was significant ($p < .05$).

In five normal subjects, the curves for the chest wall were sigmoid in the seated position but had an increasing slope in the supine position. Such curves were not observed in either the seated or supine position in any of the obese subjects.

D. Discussion

While the compliance of the lung may be studied with relative ease in the spontaneously breathing subject, that of the chest wall (i.e. those extrapulmonary structures which are deformed or displaced during breathing) is more difficult to measure. It has been estimated in the "relaxed" normal subject, ventilated by a respirator (14), and in anesthetized subjects under the influence of muscle relaxants (15), by subtracting the compliance of the lung from that of the total respiratory system. The disadvantages of these methods are the necessity for expert co-operation from the subject, or special conditions such as anesthesia and muscular paralysis.

The studies of the compliance of the respiratory system, reported in this paper, have been carried out in spontaneously breathing unanesthetized subjects. Our results in normal subjects are similar to those of Heaf and Prime, who have provided the only other estimate of the compliance of the chest wall in spontaneously breathing unanesthetized subjects (12). Our method differed from theirs in that static pressures were applied in a plethysmograph rather than at the upper airway. They have pointed out that the measurement of chest wall compliance, by the technique used

in the present study, is based on the assumption that the volume change, produced by the application of pressure around the thorax, is dependent only on the passive resistance to distension offered by the chest wall and lung. This implies that no active resistance by the respiratory musculature must be overcome. That this situation obtained, is suggested by the electromyographic studies reported in this paper, and the fact that our results also agree closely with those of Butler and Smith (15), who studied subjects under the influence of anesthesia and muscle relaxants. Furthermore, Heaf and Prime found that pressure-volume curves obtained by "relaxation pressures" and by statically applied pressures were similar.

It is assumed in the measurement of the compliance of the total respiratory system and chest wall that the pattern of distension produced by application of pressure is the same as that which occurs during normal breathing. However, to date, no evidence supporting this assumption has been forthcoming.

The finding that the pressure-volume curve for the chest wall was not sigmoid in some of the normal subjects, but had an increasing slope over the range of lung volume obtained, is of considerable interest. This phenomenon has been reported by other workers (12). Since the subjects in whom this was observed

were breathing at a low percentage of their total lung volume at rest, this phenomenon may in some way be related to resting lung volume. This is supported by the finding that this phenomenon appeared in two subjects only when they assumed the recumbent position when the resting level was reduced.

The data presented indicate that the compliance of the total respiratory system is reduced in obese individuals. This reduction in compliance is almost entirely due to a reduction in compliance of the chest wall. In other words, there is an increase in elastic resistance to distension in obese individuals. In Table VI it can be seen that if a normal efficiency is assumed, the respiratory muscles of obese individuals are performing more than twice as much work as those of normal individuals (16). One-third of this increase is due to increased elastic work done on the chest wall. The remainder of the increase must, therefore, be attributed either to invalidity of the assumption of a normal efficiency of the respiratory muscles in obese individuals, or to an increase in non-elastic work, or both. These findings support the hypothesis of other investigators, that the increased oxygen cost of breathing in obesity is, at least in part, due to an increased mechanical work done to overcome elastic resistance of the chest wall.

Table VI

MECHANICAL WORK OF THE RESPIRATORY MUSCLES IN NORMAL AND OBESE SUBJECTS

	Mechanical Work (Kg. m./l.)			
	Total Work	Elastic Work**		
		Total	Lung	Thorax
Normal	.227*	.090	.039	.051
Obese	.540+	.200	.050	.150

* mean value determined by Cherniack (16) in normal subjects.

\+ calculated from the mean O_2 cost of breathing in obese subjects determined by Kaufman et al (5) assuming a normal efficiency viz. 8.0%.

** values for elastic work calculated from mean compliance values obtained in present study.

The reduction in vital capacity in obese individuals is related to a reduction in the compliance of the total respiratory system, which is largely due to a reduced distensibility of extra-pulmonary structures. It was expected that total respiratory compliance would also be related to a measure of the size of the respiratory system, such as FRC. However, such a relationship could not be demonstrated in the present study.

It is noteworthy that the compliance of the total respiratory system is reduced even further in the recumbent position in obese individuals. The increase in resistance to distension during recumbency is entirely due to an increased resistance offered by the chest wall in this position. Thus, while the resistance of the chest wall is an average of over 70% of the total resistance to distension in the obese subject in the seated position, this is increased to over 80% in the supine position.

E. Summary

The compliance of the total respiratory system and its components was studied in 24 normal and 12 obese spontaneously breathing unanesthetized subjects. The mean compliance of the total respiratory system was .119 l./cm.H_2O in normal individuals, but was .052 l./cm.H_2O in obese subjects, indicating an increased elastic resistance to distension. The compliance of the lung in obese individuals was not different from that of the normals. The compliance of the chest wall was .224 l./cm. H_2O in normal subjects, and was .077 l./cm. H_2O in obese individuals. In contrast to normal subjects, total respiratory compliance was markedly reduced by recumbency in obese individuals. This was entirely due to a further increase in the resistance of the chest wall. A significant correlation was demonstrated between vital capacity and total respiratory compliance in normal and obese subjects. It has been estimated that one third of the increase in the mechanical work of breathing in obesity is due to elastic work done on the chest wall.

F. Acknowledgements

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