JCI The Journal of Clinical Investigation

STUDIES OF TOTAL PULMONARY CAPACITY AND ITS SUBDIVISIONS. VIII. OBSERVATIONS ON CASES OF PULMONARY FIBROSIS

Alberto Hurtado, … , W. D. W. Brooks, William S. McCann

J Clin Invest. 1935;14(1):81-93. https://doi.org/10.1172/JCI100661.

Research Article

STUDIES OF TOTAL PULMONARY CAPACITY AND ITS SUBDIVISIONS. VIII. OBSERVATIONS ON CASES OF PULMONARY FIBROSIS

By ALBERTO HURTADO, NOLAN L. KALTREIDER, WALTER W. FRAY, W. D. W. BROOKS[1] AND WILLIAM S. McCANN

(From the Department of Medicine, University of Rochester School of Medicine and Dentistry and Medical Clinic of the Strong Memorial and Rochester Municipal Hospitals, Rochester, New York)

(Received for publication September 10, 1934

The interpretation of the roentgenograph of pulmonary fibrosis in terms of functional respiratory disability is one of the most difficult problems which confronts the physician in industrial medicine. While the anatomical lesions of pulmonary fibrosis and their roentgenographic detection have been studied extensively, the mechanisms responsible for the respiratory disability are inadequately understood. The functional aspect of the fibroses acquires special importance in cases of pneumoconiosis.

Measurements of the total pulmonary capacity and its subdivisions and their relation to anatomical findings in patients with chronic pulmonary diseases have been presented in previous communications (1) (2). The limits of variation in normal subjects, both male and female, and the method for the prediction of the normal capacity of a given subject have been described (1) (2) (3). The purpose of the present communication is to correlate the anatomical findings with measurements of the total pulmonary capacity and its subdivisions in 58 cases of pulmonary fibrosis.

MATERIAL AND METHODS

Fifty-eight cases with roentgenographic evidence of pulmonary fibrosis have been studied, fifty-seven were male, and one was female (Case 51). The ages of the patients ranged from 31 to 71 years. Sixty-six per cent were below 50 years of age and thirty-one per cent were between 50 and 60 years. Only two cases were over 60 years of age.

All but four cases had been exposed to the inhalation of inorganic dusts from one to forty years. In 45 cases a history of exposure to siliceous dust in various trades (sand blasting, foundry work and several other mixed occupations) was given by the patients. Eight patients previously had been miners, and one had been a cutter of stone. In the four cases of pulmonary fibrosis with no history of industrial exposure to dust, chronic respiratory infection was the probable etiological factor. Reliable information as to the amount and composition of the dust was not available. Dyspnea, cough, loss of weight and night sweats were the most frequent symptoms. In 35 cases a fairly accurate estimation of the degree of respiratory disability could be made both from the history and from the response to exercise. In all cases dyspnea was experienced on exertion, and in three instances was caused by the slightest activity.

Clinical and electrocardiographic evidence suggestive of myocardial degeneration was occasionally encountered, but only in one instance (Case 56) was a history of cardiac decompensation obtained. There was no evidence of congestive heart failure at the time of the examination. Roentgenographic examination of the lungs, including fluoroscopy, was made in all cases. The changes detected in the roentgenographic films varied from slight accentuation of the linear markings to dense fibrotic lesions, and this has been made the basis for a grouping of our cases. In two cases pulmonary tuberculosis was strongly suspected but could not be definitely established. In 17 cases there was roentgenographic evidence of left ventricular hypertrophy; of these cases twelve occurred in the group of patients with minimal fibrotic changes, increased linear markings. Enlargement of the right ventricle was present in one patient with extensive reticular fibrosis and in one patient with minimal lung lesions; in another one with nodular fibrosis a prominent shadow in the region of the pulmonary

[1] Travelling Fellow of the Rockefeller Foundation, Fereday Fellow, St. John's College, Oxford.

artery was observed. Electrocardiograms were made in 46 cases. In 18 cases the tracings were normal, while in 16 others there was left axis deviation and of these twelve were in the group of cases with minimal roentgenographic changes. Right ventricular preponderance was found in three cases with more advanced and generalized lesions in the lung fields. In four instances myocardial damage was suspected from the electrocardiograms, and in two other cases a delay in auriculoventricular conduction without dropped beats. Only eight of all the patients examined showed clubbing of the fingers. No correlation existed between the degree of clubbing and the extent of the fibrosis as revealed by roentgenographs. The venous pressure was determined in a few instances by the method of Eyster (4). In seven patients with nodular fibrosis the venous pressure varied between 65 and 110 mm. of water with an average value of 85 mm. In four patients with a history of asthmatic attacks the venous pressure readings were 60, 90, 110 and 180 mm. of water, respectively.

The methods used for the determination of the total pulmonary capacity and its subdivisions, and for the measurement of the chest expansion have been fully presented in the communications already mentioned (1) (2) (3). Briefly summarized they consist of the determination of the residual air by the oxygen dilution method of Christie (5) and of the vital capacity and its components by graphic registration of spirometric tracings. All observations have been made at least in duplicate, and were carried out with the patients in the recumbent position after a preliminary rest of at least 15 minutes. The chest expansion was measured by means of a roentgenograph doubly exposed at a distance of six feet at maximum expiration and inspiration. The product of the area of the lung fields in the position of maximum inspiration and the anteroposterior diameter of the chest in the same position represents the "radiological chest volume." From this volume the corresponding normal total capacity and its subdivisions are predicted. Various writers have assigned different terms and values for the subdivisions of the total pulmonary capacity. We have adopted the terminology described in the first paper of this series. The normal values used for comparison were obtained from young healthy adults and consequently they may not be strictly applicable to older patients, especially since decreased vital capacity occurs in normal subjects 50 years of age or older (6). The error introduced by using such standards is, however, only relative. Investigations are now in progress, in order to establish normal values for the pulmonary capacity and its subdivisions for older individuals.

Grouping of cases on the basis of roentgenographs of the chest

One of the main objects of the present investigation was to correlate alterations in pulmonary capacity and its subdivisions with the degree of the pathological change in the lungs. The cases have, therefore, been grouped according to the nature and the extent of the roentgenographic lesions.

Group I consists of 23 cases with increased linear markings in the lung fields. Very slight feathering and beading were observed in a few instances.

Group II includes the seven cases with a history of chronic bronchial asthma. The roentgenographic findings were similar to those of Group I but in addition the shadow of the diaphragm appeared low and flat and the intercostal spaces were widened.

Group III. These 17 patients showed nodular shadows.

Group IV. In this group of four cases the nodular shadows show a tendency to agglomerate, giving a mottled appearance.

Group V. The four patients in this group presented large dense shadows, chiefly in the upper portions of the lung and in addition showed marked emphysema at the bases of the lungs.

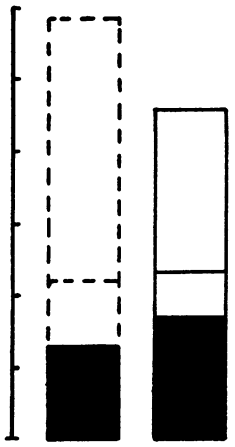
Group VI. A fine and diffuse reticular fibrosis involving the entire lung fields was present in the three cases included in this group.

Observations on the pulmonary capacity

General findings. Before considering separately the findings in each of the groups mentioned above, it will be of interest to study the alterations found in cases of pulmonary fibrosis when they are considered as a single group. The results are shown in Table I. The cases with a

TABLE I

Pulmonary capacity in 50 cases of pulmonary fibrosis *



	Mean	Standard deviation	Coefficient of variation	Variations
Absolute values				
	liters	*liters*	*per cent*	*liters*
Total Capacity	4.61 ± 0.08	0.91	19.7	2.70–7.06
Vital Capacity	2.86 ± 0.06	0.69	24.1	1.44–4.66
Mid Capacity	2.34 ± 0.08	0.91	38.8	1.26–4.80
Residual Air	1.71 ± 0.05	0.56	32.7	0.68–3.27
Complementary Air	2.26 ± 0.06	0.69	30.4	0.80–4.36
Reserve Air	0.63 ± 0.03	0.28	44.4	0.18–1.64
Relative values (total capacity = 100 per cent)				
Vital Capacity	63.0 ± 0.91	9.6	15.2	39.1–79.6
Mid Capacity	50.9 ± 1.02	10.7	21.0	30.5–81.5
Residual Air	37.2 ± 0.93	9.8	26.3	20.4–60.9
Complementary Air	49.3 ± 1.04	10.9	22.1	18.8–69.5
Reserve Air	13.3 ± 0.64	6.8	51.1	3.2–40.2

Left column represents the mean calculated normal capacity; the right column the mean observed pulmonary capacity. The black area represents the residual air; the line dividing the white area (vital capacity) is the mid capacity level.
* The cases with a history of bronchial asthma and the single observation in a female patient have been excluded from this summary.

history of bronchial asthma (Group II) and the female patient are not included. The observed mean value of the total capacity was 4.61 liters, which, compared with the calculated normal value of 5.83 liters, indicates a significant decrease in the volume of air in the lungs at maximum inspiration. This decrease in observed total capacity is caused by diminution in the vital capacity, the latter having a mean value of 2.86 liters as compared with the corresponding normal value of 4.55 liters. These changes have been found in all cases investigated. Both components of the vital capacity (the complementary and the reserve air) are equally affected. The observed residual air was moderately increased, being 1.76 liter, as compared with the calculated normal volume of 1.26 liter. The mean observed value of 2.26 liters for the mid capacity corresponded closely with the normal predicted volume of 2.21 liters.

The changes in the absolute values of the pulmonary capacity are reflected in the relative values of the subdivisions (total capacity = 100 per cent). The mean vital capacity for the entire group constitutes 63 per cent of the total capacity as compared with the normal value of 78 per cent. Correspondingly the residual air represents 37 per cent of the total capacity in pulmonary fibrosis in contrast with the normal value of 22 per cent. The relative value of the mid capacity is also increased. A graphic representation of the calculated and observed values of the pulmonary capacities and its subdivisions is given in Figure 1. The relative values are presented in Figure 2.

Findings in the different groups. A summary of the average findings in the six groups of cases is presented in Table II. A definite correlation exists between average observed values for the pulmonary capacities of the various groups and the nature and extent of the pulmonary lesions seen in the roentgenographic film. The group with minimal changes (Group I) in the roentgenographs, shows the least change in the average values of the pulmonary capacities. The total and vital capacities are moderately reduced and the ratio of residual air to total capacity is at the upper limit of normality. In Groups III and IV in which are included the cases which show nodular shadows, either uniformly or mottled, the alterations in the pulmonary capacities are more accentuated. This is chiefly due to the greater diminution in the vital capacity while the residual volume remains approximately the same. The ratio of residual volume to total capacity is definitely above the normal limits of variation. It is interesting that no significant differences in the values for the

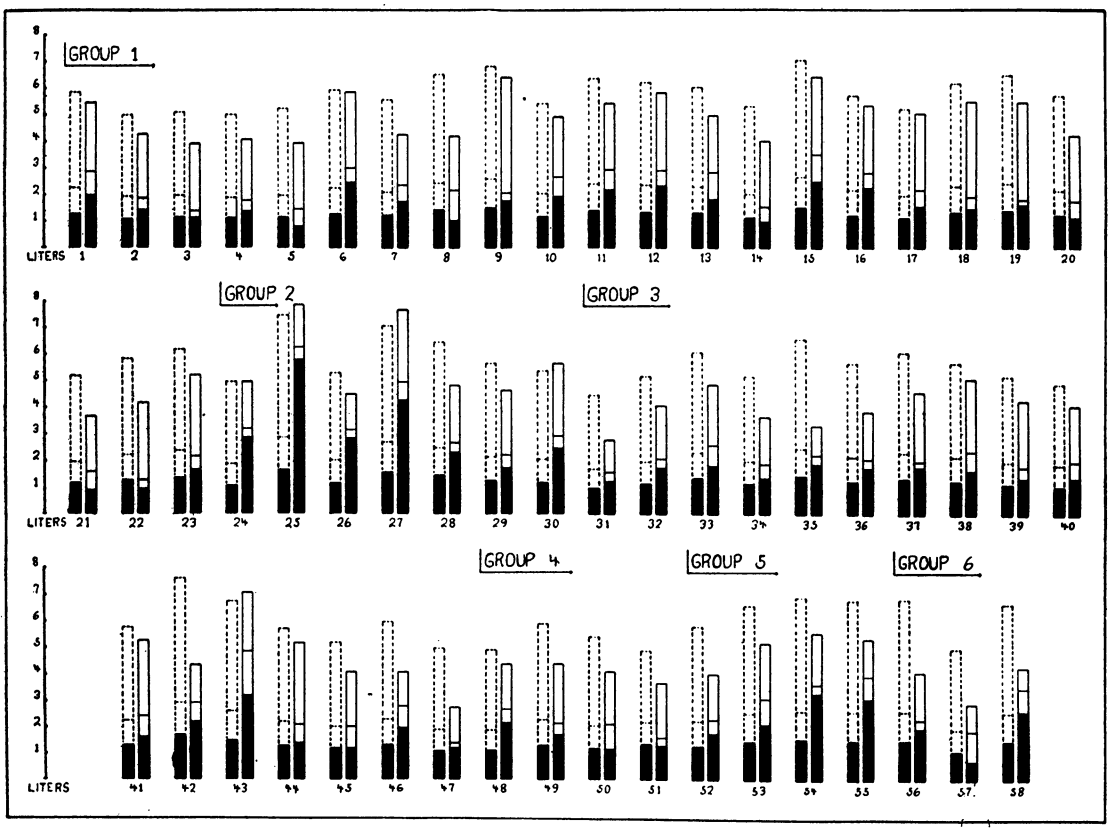


FIG. 1. CALCULATED AND OBSERVED PULMONARY CAPACITY IN 58 CASES OF PULMONARY FIBROSIS.

Each case is represented by two columns. The one on the left is the calculated normal value, and the column to the right is the observed volume. In black, the residual air. The line dividing the white area (vital capacity) is the mid capacity level. The whole column is the total capacity.

pulmonary capacity and its subdivisions exist between Groups III and IV. In Group V characterized by signs of marked emphysema at both bases and large dense shadows in the upper portions of the lungs, the relative values resemble those observed in pulmonary emphysema, except that the total capacity is reduced; although the vital capacity is less than that of the preceding groups, the absolute and relative values for the residual volume are definitely increased. The patients with a diffuse reticular fibrosis, Group VI, show the most marked reduction in vital capacity; the residual volume, however, is only moderately increased. The total capacity is markedly decreased in this group. We have reserved for final consideration the cases with a history of chronic bronchial asthma (Group II). The roentgenographic findings are similar to those observed in Group I except that evidence of pulmonary emphysema is present. The alterations in the pulmonary capacities are sharply differentiated from the rest of the cases. There is a marked decrease in the vital capacity with a proportional increase in the residual volume so that the total capacity closely approximates the normal value. The value of the ratio of residual air to total capacity is 53.9 per cent, which is abnormally high. These findings are quite similar to those previously described (6) in cases of pulmonary emphysema.

We had the opportunity to observe the changes and the development of bronchial asthma in a patient with pulmonary fibrosis, a Polish man, 51 years of age, a hard coal miner for many years. Before the beginning of asthma the usual changes of pulmonary fibrosis were observed, a decrease in the total and vital capacities with a moderate increase in the residual air (see Figure 3). About a year later this man returned to us with

the history of frequent asthmatic attacks during the interval. A second investigation revealed that the residual air had increased from the previous value of 2.09 liters to 3.80 liters, and that the vital capacity had been reduced about 0.50 liter; the total capacity at this time was greater, 6.40 liters, closely approximating the calculated value of 6.58 liters. In brief, the pulmonary capacity had changed from one typical of pulmonary fibrosis to that usually observed in emphysema.

The fact that the pulmonary capacity may be correlated in a general way with the roentgenographic appearance of the pulmonary lesions must not be interpreted as meaning that the correlation exists in each case. Very frequently a patient with minimal pulmonary fibrosis showed a more marked respiratory disability and more accentuated alterations in the pulmonary capacity than that of another patient with nodular type of fibrosis. It is not uncommon to find cases with advanced pulmonary lesions with a surprisingly good respiratory adaptation to physical activity. Study of each case is essential. A striking demonstration of this fact is found in those cases with increased residual air (Group II) who show minimal changes on the roentgenographic film, and yet exhibit marked alterations in the pulmonary capacity accompanied by a severe respiratory disability.

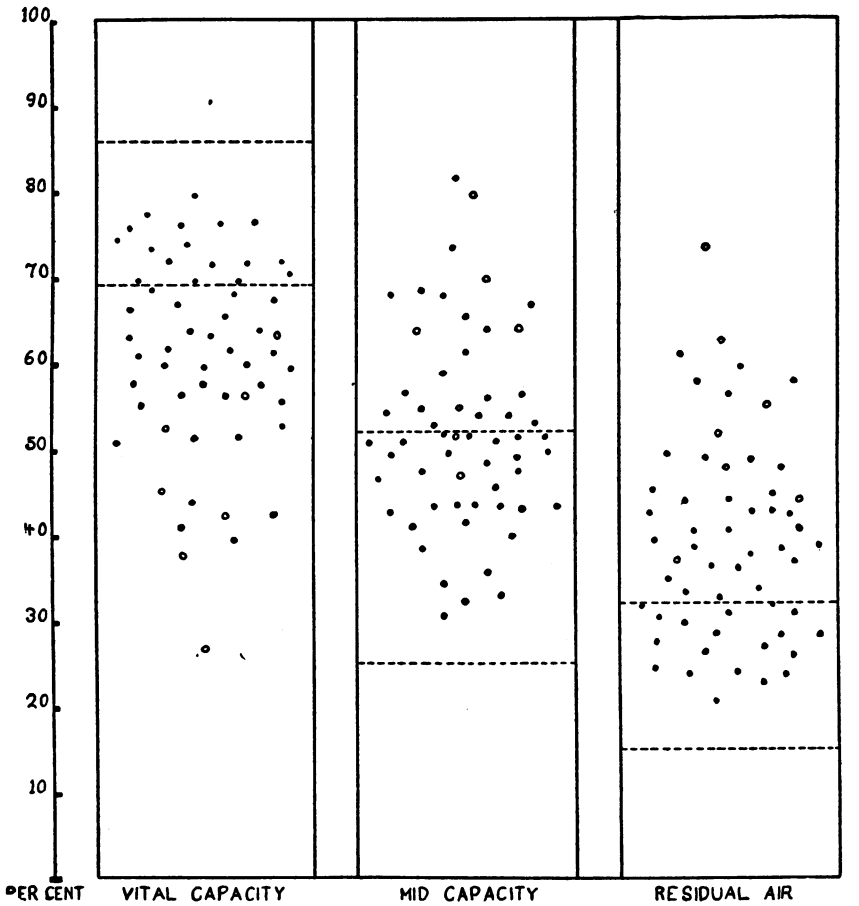


FIG. 2. RELATIVE VALUES OF THE VITAL CAPACITY, MID CAPACITY AND RESIDUAL AIR (TOTAL CAPACITY = 100 PER CENT) IN 58 CASES OF PULMONARY FIBROSIS.

Each dot represents an individual case, those with a history of asthma are represented by circles. Area between interrupted lines is zone of normal variation.

TABLE II

Average observed values of pulmonary capacity and chest expansion and their percentage differences from normal in the different groups of pulmonary fibrosis

	Group I— 23 cases		Group II— 7 cases		Group III— 17 cases		Group IV— 4 cases		Group V— 4 cases		Group VI— 3 cases	
	Observed values	Percentage difference from normal	Observed values	Percentage difference from normal	Observed values	Percentage difference from normal	Observed values	Percentage difference from normal	Observed values	Percentage difference from normal	Observed values	Percentage difference from normal
Capacities												
Total capacity, *liters*	4.90	−16.0	5.72	−5.4	4.28	−24.5	4.09	−21.9	5.00	−23.4	3.72	−39.6
Vital capacity, *liters*	3.29	−27.6	2.56	−44.8	2.62	−40.3	2.54	−36.4	2.45	−51.4	1.98	−57.4
Mid capacity, *liters*	2.19	− 1.4	3.60	+54.4	2.26	+ 3.6	2.10	+ 4.1	3.19	+27.7	2.53	+ 7.4
Residual air, *liters*	1.61	+24.9	3.16	+134.8	1.66	+31.0	1.55	+29.2	2.55	+76.1	1.74	+23.0
Complementary air, *liters*	2.71	−25.0	2.12	−42.1	2.02	−41.4	1.99	−35.7	1.81	−54.9	1.19	−68.4
Reserve air, *liters*	0.58	−37.6	0.44	−55.3	0.60	−34.4	0.55	−38.4	0.64	−40.9	0.55	−14.4
Ratios												
Vital/Total capacity × 100	67.9	−12.9	46.1	−40.9	60.8	−22.0	62.5	−19.9	49.7	−36.3	55.6	−28.7
Mid/Total capacity × 100	44.8	+18.2	61.5	+62.2	52.7	+39.0	50.9	+34.3	63.2	+66.7	67.2	+74.6
Residual/Total capacity × 100	32.1	+45.9	53.9	+145.0	39.2	+78.1	37.5	+70.4	50.3	+128.6	44.4	+101.8
Complementary/Vital capacity × 100	81.9	+ 3.1	82.8	+ 4.2	77.2	− 2.7	79.0	− 0.5	74.1	− 6.6	60.0	−24.4
Expansion												
Ratio $\frac{\text{Area at maximum expiration}}{\text{Area at maximum inspiration}} \times 100$	65.6	+ 5.3	75.4	+21.2	72.5	+16.5	69.8	+12.2	81.1	+30.4	72.5	+16.5
Right diaphragm excursion, *cm.*	4.8	−23.8	3.8	−39.7	3.9	−38.1	3.9	−38.1	2.6	−58.7	4.4	−30.1
Left diaphragm excursion, *cm.*	5.0	−21.8	4.2	−34.4	4.1	−35.9	4.1	−35.9	2.6	−60.3	3.6	−43.7
Lateral expansion, *cm.*	2.3	−28.1	2.0	−37.5	2.1	−34.3	2.5	−21.9	1.4	−56.2	1.7	−46.8
Rib rotation, *degrees*	15.0	−29.9	15.0	−29.9	13.7	−35.9	16.2	−24.3	10.0	−53.2	11.0	−48.6

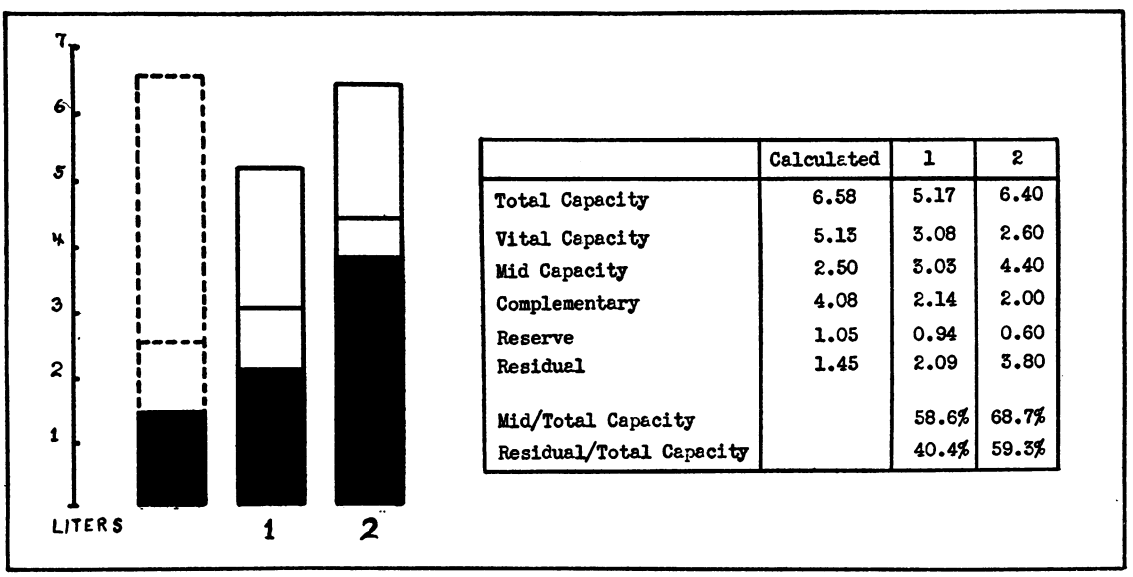


FIG. 3. PULMONARY CAPACITY IN A CASE OF PULMONARY FIBROSIS BEFORE (COLUMN 1) AND A YEAR AFTER THE DEVELOPMENT OF BRONCHIAL ASTHMA (COLUMN 2).

The column at the left with broken lines is the calculated normal capacity.

Observations on the expansion of the chest

The different roentgenographic measurements of the expansion of the chest are summarized for all cases in Table VII, and the averages for the different groups are given in Table II. There is a significant decrease in the ability to expand the chest in patients with pulmonary fibrosis. The ratio $\frac{\text{area at maximum expiration}}{\text{area at maximum inspiration}} \times 100$ has a mean value of 72.0 as compared with 62.0 in normal subjects. The diaphragms have a diminished excursion, the lateral expansion of the chest is reduced and the ribs rotate through a smaller angle in passing from the respiratory position of maximum expiration to inspiration. It is quite interesting that the average changes in chest expansion are also correlated with the nature and extent of the pulmonary lesions in the different groups. Those patients with minimal fibrosis (Group I) are able to expand their chest almost as well as normal subjects but, as the fibrotic lesions become more extensive and diffuse, the expansion of the chest is also correspondingly diminished. The patients of Group II with a history of bronchial asthma and unquestionable emphysema, and those in Group V, exhibit the greatest inability to expand the chest. It must be also emphasized that such average correlation between chest expansion and anatomical lesions, is not absolutely constant in all cases. Quite frequently a patient with marked anatomical changes shows an almost normal expansion, which again indicates the necessity of studying each case individually.

Discussion of results from the points of view of probable etiology and occupational history

It must be clearly emphasized that the consideration of our findings in regard to the pulmonary capacity from these points of view offers considerable difficulties and must be interpreted within certain limitations. The number of cases studied is inadequate to draw any statistical conclusion as to the rôle of each etiological factor, and the history given in many instances was questionable as to accuracy in regard to the exact nature and the length of exposure to dust inhalation. However, some interesting facts have been brought out.

Table III summarizes the average values and the variations of the pulmonary capacity when the cases are grouped according to their occupational history. Of those patients exposed to the inhalation of siliceous dust in various occupations, it is interesting to note that those who had worked in sandblasting gave a history of the shortest period of exposure and the shortest interval between the exposure and the development of the symptoms. They also exhibit the most marked

TABLE III

Study of 58 cases of pulmonary fibrosis from the point of view of the probable etiology and occupational history

Occupation	Number of cases	Average duration of			Percentage of cases in the various groups						Pulmonary capacities average values								
											Total capacity		Vital capacity		Mid capacity		Residual air		Ratio $\frac{\text{Residual}}{\text{Total capacity}}$
		A*	B**	C***	I	II	III	IV	V	VI	Observed values	Difference from normal	Observed values	Difference from normal	Observed values	Difference from normal	Observed values	Difference from normal	
		years			per cent						liters	per cent	liters	per cent	liters	per cent	liters	per cent	per cent
1. Sand blasting	10	4.2	3.9	6.0	20.0		60.0	20.0			4.05	−25.9	2.45	−42.4	2.18	+ 4.8	1.61	+33.1	40.1
2. Iron mouldering—Foundry work	15	17.3	15.1	22.2	46.6	20.0	20.0	6.6		6.6	5.31	−14.1	3.11	−34.0	2.84	+19.0	2.19	+56.4	39.1
3. Various mixed occupations (probably exposed to silica dust)	20	11.2	10.2	13.5	55.0	10.0	30.0		5.0		4.84	−16.2	3.02	−32.7	2.38	+ 8.0	1.81	+42.2	37.1
4. Coal mining	8	14.2	24.8	29.5	25.0	25.0	12.5		37.5		4.41	−23.7	2.42	−46.0	2.47	+11.5	1.99	+55.4	44.0
5. Stone cutting	1	40.0	39.0	41.0			100.0				4.54	−24.8	2.80	−40.6	1.94	−15.6	1.74	+30.9	38.3
6. *Unknown etiology* (? infectious)	4				25.0			25.0		50.0	4.56	−28.6	2.56	−40.0	2.20	− 2.8	1.50	+10.1	36.5

* A Exposure to dust.

**B Interval between beginning of exposure and initiation of symptoms.

***C Interval between beginning of exposure and present investigations.

alterations in the pulmonary capacity and its subdivisions; chiefly affecting the vital capacity which is markedly reduced. A high percentage of the patients in this occupation fall into the groups characterized by nodular fibrosis. Those patients who gave a history of having worked in iron foundries and various mixed occupations had a considerably longer period of exposure and have come to us at a considerably later period. The patients working in these latter occupations belong to the group of cases showing an increase of linear markings in roentgenographs.

The respiratory dead space

The volume of the respiratory dead space is of great significance in relation to the effective alveolar ventilation. An increase in the dead space relative to the tidal air tends toward a less effective alveolar ventilation. In Table IV are presented the results of the calculation of the respiratory dead space from the tidal volume and the CO_2 and O_2 percentages of the alveolar and expired air in twelve cases. The dead space has a value of about 180 cc. which compares with an average tidal volume of 0.52 liter, a figure slightly greater than the average of normal. The results of this calculation are open to objection on the grounds that it is not certain that a true average sample of alveolar air was obtained in these cases of pulmonary fibrosis. If the values obtained are correct they would indicate that an abnormality of the dead space does not play a significant rôle in the production of respiratory disability in these cases.

TABLE IV

Respiratory dead space in pulmonary fibrosis
(Calculated from the tidal volume and the CO_2 and O_2 percentages of the alveolar and expired air)

Case number	Ventilation per minute	Respirations	Tidal volume	Dead space	
				From CO_2 percentage	From O_2 percentage
	liters	per minute	liters	cc.	cc.
3	10.99	20	0.54	178	195
6	7.59	12	0.62	233	235
7	13.34	20	0.68	307	212
31	8.84	22	0.41	122	90
32	7.36	16	0.48	180	158
33	9.46	22	0.43	216	233
34	6.68	16	0.42	186	193
37	9.66	18	0.52	149	140
48	9.15	20	0.47	133	171
49	10.45	19	0.54	213	248
52	5.06	12	0.42	83	42
57	9.33	14	0.67	195	176
Average	8.99	17.6	0.52	183	174

We have attempted to reach a more accurate estimation of the dead space based upon the point at which the carbon dioxide concentration assumes a constant value in serial fractional samples of the air of a single expiration. One such curve is shown in Figure 4, in which it may be observed that the flat part of the CO_2 curve seems to be reached before 200 cc. of the air is expired, a figure which is in general agreement with that obtained in the above indirect method of calculation.

DISCUSSION

The observations presented show that there are evident alterations in the total pulmonary capacity and its subdivisions in pulmonary fibrosis. Apart from a preliminary communication from this clinic (7) there are no previous observations in the literature with which we may compare our findings. Perhaps the most closely related observations have been made in pulmonary tuberculosis. Garvin, Lundsgaard and Van Slyke (8) in 1918 observed a decreased vital capacity and increased residual air in incipient cases, while in more advanced conditions the latter volume was normal, but with a still greater reduction in the vital capacity. These findings were later confirmed by Anthony (9). It appears that a decrease in the total and vital capacities is of most constant occurrence in pulmonary fibrosis, while the residual air is as a rule moderately increased, and may infrequently reach an abnormally high value. Accompanying these alterations there is, in most cases, a decreased ability to expand the chest. These abnormalities show a definite tendency to be correlated with the degree of fibrosis.

Gardner (10) has recently summarized the development of the anatomical changes which result from the inhalation of the inorganic dust particles, and divides the reaction into five general phases: 1—diffuse parenchymatous disease due to the accumulation of phagocytes and local inflammatory changes in the immediately adjacent connective tissue; 2—linear perilymphatic proliferation; 3—

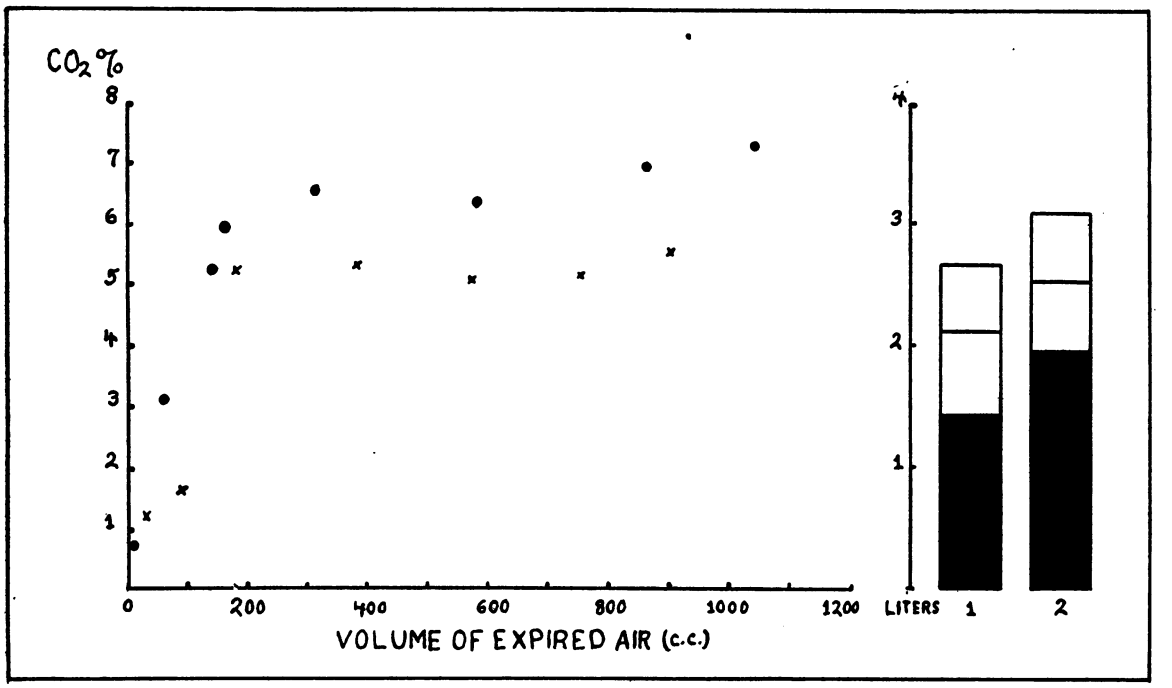


FIG. 4. CO_2 CONTENT OF VARIOUS SAMPLES TAKEN AT KNOWN VOLUMES IN A SINGLE EXPIRATION.

Black dots are samples taken in a case of pulmonary fibrosis, and crosses in a normal male subject. Column 1 represents the residual volume (black area) and the volume of the expiration (white area). The line dividing the white area is the resting respiratory level; therefore the space above this line is the tidal volume and the space below is the reserve air. Column 2 represents similar measurements in the case of pulmonary fibrosis.

beading of the trunks due to chronic proliferative changes in associated lymphoid tissues; 4—enlargement of the mediastinal lymph nodes due to proliferation of the local connective tissues, and 5—late chronic proliferation and nodule formation in the finer connective tissues of the pulmonary parenchyma. In the evolution of these pathological changes one may visualize a gradual encroachment of the fibrosis upon the functional alveolar air space, resulting in a progressive decrease in the total and vital capacities. It is somewhat more difficult to visualize the mechanism by which the residual air is increased. It is possible that as some alveoli are obliterated by fibrosis others may expand to fill the space by a compensatory emphysema. Then too, the increase in rigidity of the structure of the lung may resist the normal deflating power of its elastic tissue. Christie (11) has recently indicated that the loss of elastic properties of the alveolar wall is the factor responsible for the increase in the residual air observed in pulmonary emphysema. Binger (12) and Lundsgaard (13) explained a similar, but more moderate, increase in residual air occurring in cases of cardiac decompensation by a decrease in the elasticity of the alveolar walls. Hurtado, Kaltreider and McCann (14) observed an increase in residual air of normal men at low barometric pressure. Under the same conditions they observed intense congestion of the pulmonary capillaries of guinea pigs and suggested that this was the cause of decreased elasticity of the alveoli and resultant increase in residual air. These observations suggest that the increase in residual air in cases of pulmonary fibrosis may be explained on the basis of alterations in the elastic properties of the alveolar walls, but there is no conclusive evidence to support this hypothesis.

The intrapleural pressure was measured in three cases of our series and the results are presented in Table V. In two of these cases there was a history of asthmatic attacks and the values for pulmonary capacity were typical of emphysema. The intrapleural pressure of these subjects

TABLE V

Intrapleural pressure in three cases of pulmonary fibrosis

	Case 25*	Case 27*	Case 32†
	cm. H_2O	cm. H_2O	cm. H_2O
Quiet breathing...	−3 to +1	+0.8 to +2.5	−3 to −5
Forced inspiration	−15	−6	−3.6
Forced expiration.	+ 3	+3.8	−1 to −0.2

* Cases 25 and 27 had a history of asthmatic attacks; pulmonary capacity determinations revealed marked emphysema.

† Cases 32 showed marked nodular lesions in the roentgenographic film. Pulmonary capacity revealed a marked decrease in the vital capacity and a moderate increase in the residual air.

was positive. In the remaining case, in which the roentgenographic study of the lungs revealed very extensive nodular lesions, there was a diminished fluctuation of the pressure on forced breathing. One of the important signs of decreased elasticity, according to Christie (11), is a decrease in the reserve volume when determined after a full inspiration, as compared with the same capacity measured after an ordinary tidal inspiration. We have found this sign in only 23 (43.4 per cent) of the 53 cases in which we sought to demonstrate it. When a decrease in reserve air was noted it varied from 0.10 liter to a total disappearance of the reserve air, but there was no correlation between the degree of change and the other values in the pulmonary capacity. These observations indicate that changes in elasticity of the lungs play a part in the abnormal respiratory mechanism of pulmonary fibrosis.

Although the relative importance of the anatomical and functional factors in the pathological physiology of pulmonary fibrosis cannot be settled at the present time, it is fair to assume that both contribute to the abnormalities in the different pulmonary capacities, the determination of which may give us a quantitative index of the limits of adaptation to increased ventilatory demands. The importance of the measurement of the vital capacity has been demonstrated by Peabody (15) and his coworkers. In pathological conditions in which the residual air is increased one would expect to find conditions unfavorable to efficient alveolar ventilation. In a previous communication (6) we have indicated the close relationship, between the ratio of residual air to total capacity and the degree of respiratory disability in cases of pulmonary emphysema; the same correlation exists in pulmonary fibrosis (Figure 5, Table VI). It is difficult to evaluate the changes in

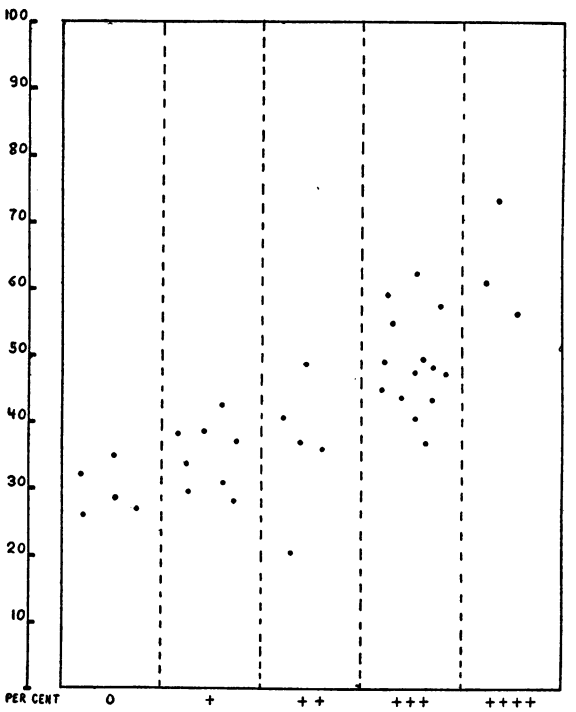


FIG. 5. CORRELATION BETWEEN THE DEGREE OF DYSPNEA AND THE RATIO $\frac{\text{RESIDUAL AIR}}{\text{TOTAL CAPACITY}} \times 100$ IN 35 CASES OF PULMONARY FIBROSIS.

0 = no dyspnea; + = dyspnea on severe physical exertion; ++ = on moderate exertion; +++ = on slight physical activity; and ++++ = dyspnea at rest.

total pulmonary capacity in relation to disability. However, from our experience with more than two hundred observations in normal subjects and in patients with chronic pulmonary disease it appears that a decrease of more than 15 per cent in the observed vital capacity is significant of abnormality; that moderate disability exists when it is 70 per cent of normal; and severe disability if it is below this limit. The ratio of residual air to total capacity, possibly abnormal when greater than 30 per cent, is definitely beyond the limits of normal if over 35 per cent, and is only moderately abnormal if not higher than 45 per cent. Beyond this limit the ratio is always associated

TABLE VI

Relation of dyspnea to pulmonary capacity

Degree of dyspnea*	Number of cases	Percentage of normal vital capacity	Percentage of normal mid capacity	Percentage of normal residual air	Ratio $\frac{\text{Mid capacity}}{\text{Total capacity}} \times 100$	Ratio $\frac{\text{Residual air}}{\text{Total capacity}} \times 100$
		per cent	per cent	per cent		
0	5	−23.1	−14.6	+ 7.3	39.1	29.6
+	8	−31.9	− 0.1	+ 31.8	46.2	35.6
++	5	−36.3	+ 6.7	+ 32.6	52.4	36.4
+++	14	−50.9	+18.8	+ 66.9	60.0	49.1
++++	3	−67.9	+48.6	+119.2	76.1	63.6

* 0 = no dyspnea; + = dyspnea on severe physical activity; ++ = on moderate exertion; +++ = on slight exertion; and ++++ = dyspnea at rest.

with a severe degree of respiratory disability and nearly always accompanied by a diminution in the saturation of the arterial blood with oxygen. Up to a certain point decrease in the vital capacity, if it is not accompanied by increase in the residual air, is compatible with a high degree of respiratory efficiency. In such a case the ratio, residual to total capacity, is not abnormally high.

It is interesting to compare the observations in pulmonary fibrosis and in pulmonary emphysema. These two conditions, apart from tuberculosis, represent the main types of chronic pulmonary disease in which respiratory disability is an important finding. Such a comparison is made in Table VII, in which we have included for comparative purposes the corresponding measurements made in normal male subjects. In patients with emphysema the chest is larger and the diaphragm is held at a lower level than in those with pulmonary fibrosis, but in both conditions the chest tends to assume a rounded shape. The ability to expand the chest is slightly less in emphysema than in fibrosis. These two conditions seem to be chiefly differentiated by changes in the total pulmonary capacities. In emphysema the total capacity is almost normal due to the great increase in the residual air which compensates for the decrease in the vital capacity. The ratio of residual volume to total capacity is higher in this disease since two factors contribute to its alteration: the decrease in the vital capacity and the increase in the residual air. On the other hand, in pul-

TABLE VII

Comparison of the mean values for chest size and expansion and pulmonary capacity in normal male subjects (50 cases) pulmonary emphysema (24 cases) and pulmonary fibrosis (58 cases)

	Normal	Emphysema	Fibrosis
Body measurements			
Body height, *cm.*	176.2±0.49	165.9±1.39	163.5±0.75
Body weight, *kgm.*	72.5±1.07	61.6± 1.82	66.6±1.07
Body surface area, *cm.²*	187.8±1.19	167.4± 2.88	172.2±1.58
Size and shape of chest			
Radiological chest volume, inspiration, *liters*	14.83±0.17	14.77± 0.76	13.86±0.22
Area of lung fields, inspiration, *cm.²*	68.5 ±7.28	630 ±12.52	603 ±7.72
Height of right diaphragm, inspiration, *cm.*	23.6 ±0.20	24.2± 0.38	21.8 ±0.27
Height of left diaphragm, inspiration, *cm.*	25.6 ±0.23	26.1± 0.36	23.8 ±0.23
Width of chest, inspiration, *cm.*	30.3 ±0.16	28.3± 0.44	29.4 ±0.22
Depth of chest, inspiration, *cm.*	21.7 ±0.18	23.1± 0.38	23.4 ±0.22
Chest index $\frac{\text{Depth}}{\text{Width}} \times 100$	68.5 ±0.55	79.3± 1.47	77.1 ±1.10
Chest expansion			
Anteroposterior expansion, *cm.*	3.9 ±0.08	2.1± 0.12	1.9 ±0.07
Lateral expansion, *cm.*	3.2 ±0.08	2.4± 0.13	2.2 ±0.08
Excursion right diaphragm, *cm.*	6.3 ±0.11	3.6± 0.22	4.2 ±0.17
Excursion left diaphragm, *cm.*	6.4 ±0.12	4.4± 0.21	4.4 ±0.17
Rib rotation, *degrees*	21.4 ±0.51	16.0± 0.79	14.4 ±0.43
Ratio $\frac{\text{Area at maximum expiration}}{\text{Area at maximum inspiration}} \times 100$	62.2 ±0.42	73.4± 1.20	70.0 ±0.87

	Normal	Emphysema	Fibrosis
Pulmonary capacity			
Absolute values			
Total capacity, *liters*	6.13±0.08	5.73±0.14	4.61±0.08
Vital capacity, *liters*	4.78±0.06	2.88±0.13	2.86±0.06
Mid capacity, *liters*	2.34±0.05	3.68±0.14	2.34±0.08
Residual air, *liters*	1.36±0.04	2.84±0.14	1.71±0.05
Complementary air, *liters*	3.79±0.05	2.04±0.08	2.26±0.06
Reserve air, *liters*	0.98±0.02	0.84±0.03	0.63±0.03
Relative values			
Vital/Total capacity × 100	78.0±0.41	50.4±1.49	63.0±0.91
Mid/Total capacity × 100	37.9±0.75	64.0±1.52	50.9±1.02
Residual/Total capacity × 100	22.0±0.41	49.6±1.52	37.2±0.93
Complementary/Total capacity × 100	61.9±0.51	35.6±1.46	49.3±1.04
Reserve/Total capacity × 100	16.2±0.39	14.6±0.58	13.3±0.64
Complementary/Vital capacity × 100	79.4±0.50	70.8±1.37	78.2±0.97
Reserve/Vital capacity × 100	20.6±0.52	29.2±1.36	21.8±0.99

The average ages of the different groups were as follows: Normal subjects, 23 years; pulmonary emphysema, 46 years; and pulmonary fibrosis, 47 years.

monary fibrosis, the total capacity is diminished. This is due to a marked decrease in the vital capacity while the residual air is only moderately increased; consequently the ratio $\frac{\text{residual air}}{\text{total capacity}} \times 100$ is not as high as in emphysema. It is also interesting to notice that in emphysema the reduction in vital capacity occurs chiefly at the expense of the complementary air, while in fibrosis complementary and reserve air are about equally reduced. It has been our experience that the respiratory disability is also of greater severity in emphysema as compared with pulmonary fibrosis. Cases of pulmonary fibrosis which have developed emphysema are, as a rule, severely disabled.

SUMMARY AND CONCLUSIONS

Determinations of total pulmonary capacity and its subdivisions and measurements of the expansion of the chest have been made in 58 cases of pulmonary fibrosis, all but 4 of which gave a history of employment in dusty trades. Additional observations included a complete clinical examination, roentgenographic and electrocardiographic studies. The observations have been correlated with the degree of the anatomical changes in the lungs revealed by roentgenographs, with the probable etiological factor, and with the degree of respiratory disability. A comparison has also been made with previous observations from cases of pulmonary emphysema. These investigations lead to the following conclusions:

1. Definite alterations in the pulmonary capacity occur in cases of pulmonary fibrosis, consisting chiefly in moderately reduced total capacity, a more marked decrease in the vital capacity and a moderate or marked increase in the residual air.

2. The above changes are usually, but not always, proportional to the nature and extent of the pathological changes in the roentgenographs of the lungs. Frequent exceptions to this correlation indicate the necessity for studying and interpreting each case individually.

3. The changes in the pulmonary capacity are found to be correlated with the degree of clinical respiratory disability. The estimation of the vital capacity and of the ratio $\frac{\text{residual air}}{\text{total capacity}} \times 100$ are important indices of respiratory efficiency; the lower the former and the higher the latter, the greater the disability.

4. No significant alteration in the volume of the respiratory dead space was found in 12 cases of pulmonary fibrosis.

5. There is a reduction in the ability to expand the chest in pulmonary fibrosis, usually, but not always, proportional to the degree of the anatomical changes revealed in roentgenographs of the chest.

6. Comparison of the pulmonary capacities in uncomplicated cases of pulmonary fibrosis and in emphysema reveal significant differences. In uncomplicated fibrosis the total capacity and vital capacity are reduced, and the residual air is moderately increased. In emphysema the total capacity is normal, due to the fact that the increase in residual air compensates for the decrease in vital capacity.

BIBLIOGRAPHY

1. Hurtado, A., and Boller, C., Studies of total pulmonary capacity and its subdivisions. I. Normal, absolute and relative values. J. Clin. Invest., 1933, **12**, 793.
2. Hurtado, A., and Fray, W. W., Studies of total pulmonary capacity and its subdivisions. II. Correlation with physical and radiological measurements. J. Clin. Invest., 1933, **12**, 807.
3. Hurtado, A., Fray, W. W., Kaltreider, N. L., and Brooks, W. D. W., Studies of total pulmonary capacity and its subdivisions. V. Normal values in female subjects. J. Clin. Invest., 1934, **13**, 169.
4. Eyster, J. A. E., The Clinical Aspects of Venous Pressure. The Macmillan Co., New York, 1929.
5. Christie, R. V., The lung volume and its subdivisions. I. Methods of measurements. J. Clin. Invest., 1932, **11**, 1099.
6. Hurtado, A., Kaltreider, N. L., Fray, W. W., Brooks, W. D. W., and McCann, Wm. S., Studies of total pulmonary capacity and its subdivisions VI. Observations on cases of obstructive pulmonary emphysema. J. Clin. Invest., 1934, **13**, 1027.
7. Hurtado, A., Fray, W. W., and McCann, Wm. S., Studies of total pulmonary capacity and its subdivisions. IV. Preliminary observations on cases of pulmonary emphysema and pneumoconiosis. J. Clin. Invest., 1933, **12**, 833.
8. Garvin, A., Lundsgaard, C., and Van Slyke, D. D., Studies of lung volume. II. Tuberculous men. J. Exper. Med., 1918, **27**, 87.
9. Anthony, A. J., Untersuchungen über Lungenvolumina und Lungenventilation. Deutsches Arch. f. klin. Med., 1930, **167**, 129.

10. Gardner, L. U., The pathologic reaction in various pneumonoconioses. J. A. M. A., 1933, **101**, 594.

11. Christie, R. V., The elastic properties of the emphysematous lung and their clinical significance. J. Clin. Invest., 1934, **13**, 295.

12. Binger, C. A. L., The lung volume in heart disease. J. Exper. Med., 1923, **38**, 445.

13. Lundsgaard, C., Determination and interpretation of changes in lung volumes in certain heart lesions. J. A. M. A., 1923, **80**, 163.

14. Hurtado, A., Kaltreider, N. L., and McCann, Wm. S., Respiratory response to anoxemia. Am. J. Physiol., 1934, **109**, 626.

15. Sturgis, C. C., Peabody, F. W., Hall, F. C., and Fremont-Smith, F., Jr., Clinical studies on the respiration. VIII. The relation of dyspnea to the maximum minute volume of pulmonary ventilation. Arch. Int. Med., 1922, **29**, 236.