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Research Article



# VENTILATORY MECHANICS IN PULMONARY EDEMA IN MAN [1]

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The mechanics of breathing have recently been investigated in patients with a variety of pulmonary abnormalities including pulmonary congestion due to heart disease (1–3). The present report describes ventilatory mechanics in eight patients with frank pulmonary edema. The data were calculated from simultaneous oscillographic records of tidal volume, air-flow, and esophageal pressure.

## CLINICAL MATERIAL

Eight patients were studied while in pulmonary edema, and seven of the eight patients were restudied following recovery from pulmonary edema. The clinical criteria for the diagnosis of pulmonary edema were as follows: 1) moderate to marked dyspnea at rest, 2) moist rales over at least one-half of both lung fields in the absence of fever or other signs of pulmonary infection, 3) evidence of incapacitating heart disease with previous episodes of pulmonary congestion or edema, 4) roentgenographic changes consistent with those seen in pulmonary edema where chest roentgenograms were available. In all cases except Case 7, rales were still present during the recovery study indicating the presence of pulmonary congestion. Three patients had rheumatic valvular heart disease, three had arteriosclerotic heart disease, one had hypertensive cardiovascular disease and the remaining patient had myocardial fibrosis and abscess formation and severe anemia. Only in Case 2 was there evidence for any other lung disease than pulmonary edema and congestion. A brief clinical account of each patient is presented in the Appendix.

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## METHODS

Intraesophageal pressure was recorded by means of a 60 cm. long polyethylene tube with an internal diameter of 0.09 cm.[5] filled with boiled water and attached to a pressure transducer of low volume displacement.[6] This system gave a 95 per cent response to a square wave within 0.02 seconds. The natural frequency was 17 cycles per second. Care was taken that the tip of the esophageal tube was in the middle or upper third of the esophagus. The fluid-filled tube was used instead of an air-filled balloon because its small size enabled it to be swallowed more easily by acutely dyspneic patients. Air-flow was measured by recording the differential pressure[7] across a 400 mesh, monel-metal screen 2 inches in diameter. Tidal volume was measured in a carefully balanced spirometer.[8] To the spirometer counter-weight was attached an electrical contact to a resistance wire which formed two arms of a resistance bridge. This bridge was activated by a mercury cell and the output fed into a DC amplifier. The output of this volume transducer was linearly related to volume up to the capacity of the spirometer bell (9 liters). Rebreathing of expired air occurred in using this spirometer. Pressure at the mouth was recorded by another pressure transducer,[9] and for normal breathing was consistently under 1.5 cm. of water. This pressure, representing the frictional and inertial resistance of the pneumotachometer and spirometer, was subtracted from esophageal pressure in calculating resistance. Esophageal pressure, air-flow, tidal volume, and mouth pressure tracings were recorded photographically as shown in Figure 1.

Lung compliance, expressed in liters per cm. of water (L. per cm. $H_2O$), was obtained by dividing the tidal volume by the change in the esophageal pressure between points of zero flow. Resistance was calculated at points of 1 L. per second air-flow by the method of von Neergaard and Wirz (4). In this method, the volume (above end-tidal volume), measured at the point at which resistance was to be determined, was divided by the compliance to yield the pressure which would be present in the esophagus if no air were flowing. The difference between this calculated "static pressure" and the pressure actually measured is the additional pressure necessary to overcome the resistance of airways, tissues

[5] Clay-Adams Co., PE 100.

[6] Statham P23 Ga pressure transducer.

[7] Statham P5 0.2 p.s.i.d. pressure transducer.

[8] Warren E. Collins Co. respirometer.

[9] Statham P23 D pressure transducer.

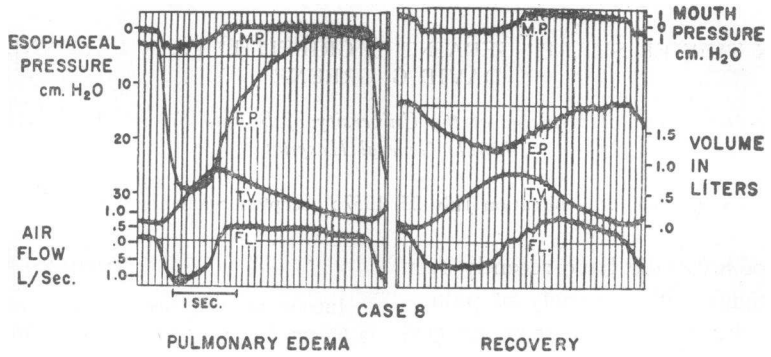


FIG. 1. OSCILLOGRAPHIC RECORD OF MOUTH PRESSURE (M.P.), ESOPHAGEAL PRESSURE (E.P.), TIDAL VOLUME (T.V.) AND AIR-FLOW (FL.) DURING PULMONARY EDEMA AND FOLLOWING RECOVERY

and apparatus. Mouth pressure (apparatus resistance) is then subtracted, leaving airway and tissue resistance. Assumption of a linear static volume-pressure relationship for the lung is necessary in order for these calculations of resistance to be valid. Ten of 20 breaths from each study were analyzed for compliance and resistance calculations.

In Cases 1, 4, 6 and 7, air-flow rose above 1 L. per second early in inspiration and fell below it again late in inspiration, so that resistance at 1 L. per second flow could be calculated at a point early in inspiration and at a second point late in inspiration. These early and late inspiratory resistances are compared in Table II and are averaged to give the single value for inspiratory resistance shown in Table I. In the remaining cases air-flow reached or exceeded 1 L. per second for only a short period during the middle of inspiration. In these cases inspiratory resistance was calculated at a single point.

Resistance has also been calculated at intervals throughout the respiratory cycle and plotted against time as in Figure 2. The resistances shown have been calculated at varying rates of air-flow by dividing the calculated resistance pressure gradient by flow. The peak resistance values obtained in early inspiration were also measured at air-flow rates other than 1 L. per second.

## RESULTS

### *Compliance*

As can be seen from Table I, compliance was found to be extremely low in pulmonary edema. The average value was 0.037 L. per cm. $H_2O$ with values ranging from 0.017 to 0.057 L. per cm. $H_2O$. These values for compliance are generally considerably lower than those reported in patients with pulmonary congestion due to heart disease. Following recovery from pulmonary edema the average compliance was 0.089 L. per cm. $H_2O$ with values ranging from 0.044 to 0.190 L. per cm. $H_2O$. These values for compliance correspond to values reported by others in patients with pulmonary congestion, and there was clinical evidence of pulmonary congestion in all but Case 7 at the time of follow-up study. All values at the time of frank pulmonary edema are considerably below the average normal compliance of 0.165 L. per cm. $H_2O$ (5). It will be noted that except in Case 6, compliance more than doubled on recovery from pulmonary edema. The values of the standard errors indicate that all differences in compliance in individual patients between pulmonary edema values and recovery values are highly significant ($p < 0.01$).

It is known that in the presence of airway obstruction, compliance falls as the respiratory rate rises (6). The average respiratory rates of the group during pulmonary edema and following recovery, respectively, were 26 and 22. The greatest individual change was from 25 to 19 breaths per minute. It is unlikely that such small changes in breathing frequency could account for compliance change of the magnitude measured.

### *Resistance*

Resistance was significantly increased in pulmonary edema averaging 9.9 cm. $H_2O$ per L. per second during inspiration and 8.7 cm. $H_2O$ per L. per second during expiration. These values are three to four times the normal. Following recovery from pulmonary edema the average resistance during inspiration was found to be 5.3 cm. $H_2O$ per L. per second and during expiration 4.4 cm. $H_2O$ per L. per second. These values are also elevated and are similar to resistance values reported by others (1) in patients with pulmonary

TABLE I

*Ventilatory mechanics in eight patients with pulmonary edema*

| Case no. | Compliance ± S.E. (L./cm. $H_2O$) | | Resistance at 1 L./sec. ± S.E. (cm. $H_2O$/L./sec.) | | | | Mean tidal volume (L. B.T.P.S.) | | Minute volume (L./min. B.T.P.S.) | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Inspiratory | | Expiratory | | | | | |
| | Pulmonary edema | Recovery | Pulmonary edema | Recovery | Pulmonary edema | Recovery | Pulmonary edema | Recovery | Pulmonary edema | Recovery |
| 1 | 0.017 ± 0.0002 | 0.044 ± 0.001 | 10.3 ± 1.0 | 9.2 ± 0.9 | 6.7 ± 1.3 | 3.6 ± 0.4 | 0.43 | 0.73 | 10.4 | 16.8 |
| 2 | 0.020 ± 0.0006 | 0.053 ± 0.003 | 12.7 ± 0.9 | 3.4 ± 0.3 | 8.9 ± 0.6 | 4.4 ± 0.3 | 0.55 | 0.38 | 17.7 | 9.9 |
| 3 | 0.026 ± 0.002 | 0.069 ± 0.004 | 12.1 ± 1.0 | 9.3 ± 0.9 | 13.3 ± 1.8 | 9.1 ± 0.8 | 0.69 | 0.74 | 16.9 | 13.8 |
| 4 | 0.031 ± 0.0007 | 0.076 ± 0.0002 | 9.1 ± 0.5 | 2.6 ± 0.2 | 3.9 ± 0.3 | 3.4 ± 0.2 | 0.64 | 1.42 | 17.9 | 31.5 |
| 5 | 0.035 ± 0.001 | | 5.9 ± 0.4 | | 11.5 ± 1.2 | | 0.54 | | 15.7 | |
| 6 | 0.052 ± 0.001 | 0.071 ± 0.001 | 11.6 ± 1.1 | 6.8 ± 0.2 | 11.4 ± 0.7 | 7.1 ± 0.5 | 0.82 | 0.85 | 18.1 | 21.3 |
| 7 | 0.054 ± 0.0005 | 0.190 ± 0.006 | 7.5 ± 0.6 | 2.7 ± 0.3 | 2.8 ± 0.3 | 0.6 ± 0.08 | 0.97 | 0.50 | 24.5 | 9.7 |
| 8 | 0.057 ± 0.001 | 0.120 ± 0.003 | 10.1 ± 0.3 | 2.8 ± 0.1 | 11.1 ± 0.6 | 2.4 ± 0.2 | 0.95 | 0.84 | 20.0 | 16.0 |
| Averages ± S.E. | 0.037 ± 0.0056 | 0.089 ± 0.019 | 9.9 ± 0.83 | 5.3 ± 1.17 | 8.7 ± 1.36 | 4.4 ± 1.09 | | | | |
| p | <0.05 | | <0.01 | | <0.05 | | | | | |

TABLE II

*Difference between early inspiratory resistance and late inspiratory resistance in four patients*

| Case no. | Resistance in cm. $H_2O$ at 1 L./sec. flow ± S.E. | | |
|---|---|---|---|
| | Early inspiration | Late inspiration | p |
| | During pulmonary edema | | |
| 1 | 13.7 ± 1.2 | 6.9 ± 0.6 | <0.01 |
| 4 | 10.4 ± 0.7 | 7.7 ± 0.5 | <0.01 |
| 6 | 16.2 ± 0.3 | 7.1 ± 0.3 | <0.01 |
| 7 | 9.7 ± 0.4 | 5.2 ± 0.2 | <0.01 |
| | Following recovery | | |
| 1 | 12.1 ± 0.9 | 6.2 ± 0.8 | <0.01 |
| 4 | 2.4 ± 0.3 | 2.7 ± 0.3 | >0.1 |
| 6 | 7.4 ± 0.3 | 6.2 ± 0.3 | <0.01 |
| 7 | 3.8 ± 0.3 | 1.6 ± 0.1 | <0.01 |

congestion. In individual cases differences in resistance between pulmonary edema and recovery are significant except for inspiratory resistance in Cases 1 and 3. The differences between the means of pulmonary edema data and recovery data are significant for inspiratory resistance ($p < 0.01$) and for expiratory resistance ($p < 0.05$).

Early inspiratory resistance was consistently and significantly greater than late inspiratory resistance during pulmonary edema in Cases 1, 4, 6 and 7 and, except in Case 4, following recovery from pulmonary edema (Table II). Figure 2 shows resistance plotted against time throughout the respiratory cycle in Case 6. It is seen that inspiratory resistance is highest early in inspiration and falls significantly late in inspiration. The upper curve shows resistance in acute pulmonary

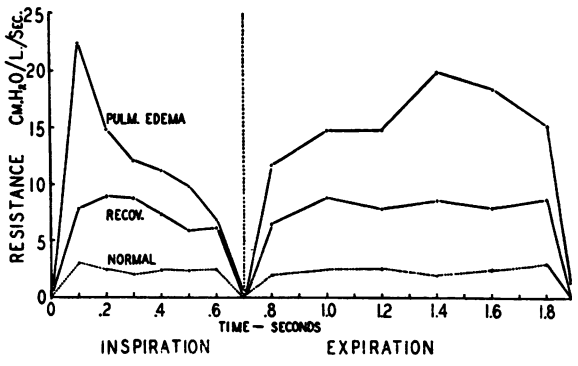


FIG. 2. RESISTANCE PLOTTED AGAINST TIME THROUGH ONE RESPIRATORY CYCLE IN CASE 6

The top curve was obtained during pulmonary edema, the middle one following recovery in the same patient (pulmonary congestion persisted), and the bottom curve from a normal subject.

edema; the middle curve, resistance following recovery when pulmonary congestion was still present; and the lower dotted curve, similar data on a normal subject. In all cases, a peak of resistance was found within the first 0.1 to 0.2 seconds of inspiration. This early inspiratory peak resistance varied from 9.1 to 31.5 cm. of water per L. per second.

The patients studied represent several etiologic types of heart disease. No characteristic relationship between compliance and resistance changes can be associated with the etiologic types of heart disease represented. An attempt was made to determine whether the ratio between compliance change and resistance change is different in patients treated with aminophyllin, a known bronchodilator on the one hand, and morphine, a known bronchoconstrictor, on the other. Three patients (Cases 4, 5 and 7) had neither of these drugs before study, one patient had aminophyllin alone (Case 6), two patients had morphine alone (Cases 2 and 8), and two patients had aminophyllin and morphine (Cases 1 and 3). The small number of patients and the several combinations of treatment limit conclusions concerning the effect of prior treatment with these agents on initial compliance and resistance. However, the absence of outstanding difference between the three cases who had not been treated with these agents prior to study and the remaining cases suggests that any direct effect on lung mechanics which these drugs may have had was a minor one. In all probability the high resistance is a function of pulmonary edema rather than the activity of drugs which are active on bronchial musculature.

## DISCUSSION

The mean compliance of the patients in this study during pulmonary edema was 22 per cent of the normal of 0.165 L. per cm. $H_2O$ as reported by Frank, Mead, Siebens, and Storey (5). Compliance averaged 54 per cent of normal following recovery. The occurrence of comparably low values for compliance in experimental pulmonary edema in the dog (7–9) is well documented. That such low compliance values cannot be accounted for by simple replacement of alveolar volume by edema fluid is indicated by simultaneous compliance and lung volume determinations done by Cook, Schreiner, and Mead (9) in dogs with pulmonary edema. These investigators showed that decreases of compliance of 70 per cent were accompanied by decreases in total and ventilated pulmonary gas volumes of only 20 and 32 per cent, respectively. Pulmonary hypertension does not account for such low compliance values as shown by the work of Frank, Radford, and Whittenberger (10) and by the observations of Borst and co-workers (11) where the fall in compliance resulting from left atrial pressures of 50 cm. of water was of the order of 20 to 30 per cent.

An alternative explanation is that a change in either the composition or the geometry of distensible elements of the lungs has occurred to render them less compliant. The principal distensible elements of the alveoli are the elastic fiber network and the alveolar surface film. It is possible that pulmonary edema alters either the properties of elastic fibers or their geometric arrangement. It appears more plausible to the authors, however, that it is the composition and the geometry of the alveolar surface film which is altered in pulmonary edema. The observations of Mead, Whittenberger, and Radford (12) and of Clements (13) indicate that alveolar surface forces play a dominant role in determining lung compliance. Observations of Brown (14) on the surface tension of pulmonary edema fluid suggest that its surface properties are much the same as those of the normal alveolar surface. However if this edema fluid were geometrically arranged as bubbles, the radii of which were smaller than those of the parent alveoli, the resulting compliance would be that of alveoli with radii equal to those of the bubbles.

Assuming that both an alveolus and its contained bubbles are portions of spheres, the volume of such a unit is approximately equal to $4\pi r^3/3$. If one considers only surface compliance, Laplace's law states that transmural pressure is directly proportional to surface tension and is inversely proportional to first power of the radius ($P = 2T/r$). Thus V/P or compliance equals $4\pi r^3/3 \div 2T/r$ or $2\pi r^4/3T$. By application of differential calculus it may be shown that the slope of the volume-pressure curve (dV/dP) as well as the compliance (V/P) varies directly as the fourth power of the radius and inversely as the surface tension. Thus halving the radius would

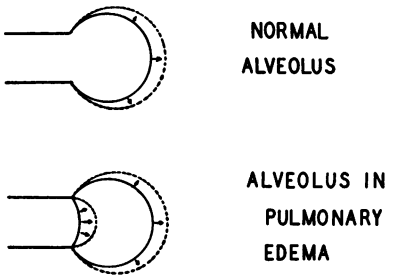


FIG. 3. DIAGRAMMATIC REPRESENTATION OF A NORMAL ALVEOLUS (ABOVE) AND AN ALVEOLUS DURING PULMONARY EDEMA (BELOW) EXPANDING DURING INSPIRATION

The bottom alveolus has at its mouth a bubble of small radius and low compliance.

decrease compliance sixteenfold, while doubling surface tension would only halve compliance. It has been suggested to us by Dr. Elwyn S. Brown that if the majority of alveoli had edema fluid bubbles at their mouths as shown in Figure 3, with inward inspiratory expansion of such bubbles the lung would show the following properties: 1) a striking decrease in compliance would occur accompanied by relatively little decrease in pulmonary gas volume; 2) the ventilated pulmonary gas volume would be less than the total pulmonary gas volume because of trapping of air distal to the bubbles; 3) compliance would be low at the beginning of inflation of the bubble and would rise as the bubbles and their radii of curvature became larger. The work of Cook, Schreiner, and Mead (9) cited above gives evidence that the first two of these effects actually occur in pulmonary edema in the dog. The third effect is also a characteristic of experimental pulmonary edema in the dog as indicated by previous work of the present authors (7) and the work of Whittenberger and Affeldt (15) who have described alinear volume-pressure curves of the lung and thorax similar to that in the right hand portion of Figure 4 in dogs with pulmonary edema.

If one considers the normal resistance not to be above 3 cm. $H_2O$ per L. per second, resistance in pulmonary edema averages more than three times the normal and following recovery averages twice the normal. This increase in resistance is not so difficult to account for as the decrease in compliance considered above. Edema of the airways could account for much of this when one considers that the resistance to laminar flow offered by a tube is inversely proportional to the fourth power of its radius. In addition, foam and secretion in the airways might increase the proportion of turbulent flow under which state the resistance is proportional to the square rather than a first power of flow. These factors would tend to make resistance highest during expiration. This is a prominent feature of resistance-time plots in several of the patients as exemplified in Figure 2. However the high value for resistance seen early in inspiration is not explained fully by a consideration of these factors. One may question whether this inspiratory peak resistance in fact represents true resistance at all. It should be recalled that this resistance is a calculated value and that its calculation involves the assumption that the whole volume-pressure curve within the range of the tidal volume is linear. The lung volume-pressure loops in Figure 4 graphically portray the two situations which might apply. The diagonals within the loops represent the static volume-pressure curves. In order to calculate resistance one assumes the relationship in the left half of the figure in which the static volume-pressure curve is linear throughout the tidal range. The right hand figure is based on the assumption that the actual volume-pressure curve may be curvilinear becoming steeper as inflation continues, as seen in experimental pulmonary edema and as discussed in the preceding paragraph. Both compliance and resistance are lower in the right hand figure at the point early in inspiration at which resistance is being measured. However, the compliances for the two figures are equal when measured over the whole tidal volume.

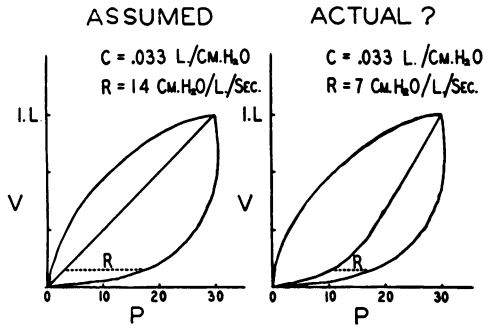


FIG. 4. TOTAL VOLUME-PRESSURE "LOOPS" DURING ONE TIDAL BREATH

The diagonal lines bisecting the loops are the static volume-pressure (compliance) curves, and the right hand limb of each loop is the dynamic inspiratory pressure-volume curve. See text for discussion.

Consequently, overestimation of resistance would occur early in inspiration. Thus it is possible that the same explanation postulated to explain the extremely low compliance and the particular alinear configuration of the lung's static volume-pressure curve in pulmonary edema may also explain the apparent high early inspiratory resistance.

The concept of critical opening pressures of terminal lung units provides another possible explanation of the physical properties of the lungs in pulmonary edema. Burton (16) has pointed out that fine tubular structures such as arterioles are fundamentally unstable and tend to close off if the balance of forces about them is even slightly altered. If tissue edema and fluid in airways narrowed and disturbed the balance of forces about respiratory bronchioles and alveolar ducts causing them to close off, fewer expandable alveoli would be available and compliance would fall. Moreover compliance would be lowest at the beginning of inspiration and would rise progressively with lung inflation as the opening pressures of more and more lung units were exceeded. The apparent high resistance early in inspiration may be a manifestation of high opening pressures of terminal lung units.

A final factor which might account for an additional decrement of compliance is based upon the knowledge that the lung and presumably its individual alveoli become less compliant as they are overdistended. In pulmonary edema, both the uncompliant or fluid-filled alveoli and the normally compliant alveoli are subjected to a greater than normal inflation pressure (greater negative intrapleural pressure). This may overdistend the more compliant alveoli so that they too are less compliant because they are operating over less compliant portions of their volume-pressure curves.

## SUMMARY AND CONCLUSIONS

1. Pulmonary compliance and resistance have been studied in eight patients in pulmonary edema and in seven of the eight following recovery from pulmonary edema.

2. Compliance was found to be very low in pulmonary edema, averaging $0.037 \pm 0.0056$ (S.E.) L. per cm. $H_2O$ or 22 per cent of the average normal value. Following recovery, the average compliance was $0.089 \pm 0.019$ L. per cm. $H_2O$.

3. Resistance was found to be markedly increased, averaging $9.9 \pm 0.83$ (S.E.) cm. $H_2O$ per L. per second (inspiration) during pulmonary edema, a value over three times the normal. On recovery inspiratory resistance fell to an average of $5.3 \pm 1.17$ cm. $H_2O$ per L. per second.

4. The highest resistance values were found early in inspiration during pulmonary edema. This high early inspiratory resistance may be an indirect expression of alinearity of the static volume-pressure curve of the lung in pulmonary edema rather than true resistance.

5. The effect on compliance of bubbles in alveoli is discussed. It is suggested that the presence of such bubbles at alveolar orifices might account for the disproportionate lowering of compliance with respect to pulmonary gas volume change, the trapping of gas, the alinearity of the static volume-pressure curve and the apparent high early inspiratory resistance found in pulmonary edema.

6. Elevation of the opening pressures of terminal lung units has been suggested as an alternative explanation for the properties of the lungs in pulmonary edema.

## APPENDIX

*Case 1,* A.F., was a 64 year old white female with inactive rheumatic valvular heart disease with both mitral and aortic involvement. Pulmonary edema was precipitated by sodium retention secondary to phenylbutazone which had been given for a tendonitis. When initially studied, moist rales were heard to the level of midscapulae bilaterally, and expiratory wheezes were prominent. The patient was considered to be in acute pulmonary edema. Before study, morphine, mercuhydrin, aminophyllin, oxygen by mask, and tourniquets had been administered to the patient. This patient was studied while in acute pulmonary edema and after recovery two days later.

*Case 2,* W. C., was a 67 year old white male with a profound macrocytic anemia of unknown cause. Moist rales were heard diffusely over both lung fields and the patient was considered to be in acute pulmonary edema. Morphine had been given approximately three hours previous to the initial study. He was studied while in acute pulmonary edema and again following recovery, four days later. Ten days following the last study the patient expired, and at necropsy in addition to severe recurrent pulmonary edema with myocardial abscesses and fibrosis, he also had pulmonary fibrosis due to silicosis. The low compliance was probably partially due to the silicosis, but it is thought that the changes in compliance and resistance which occurred with recovery represented changes in the degree of pulmonary edema.

*Case 3,* M.O., was a 54 year old white female with chronic rheumatic heart disease with mitral stenosis. When initially studied, moist rales were present from apices to bases bilaterally, and the patient was considered to be in acute pulmonary edema. The patient had received morphine and aminophyllin 80 minutes prior to the initial study and was on maintenance digitalis. When restudied two days later she was not dyspneic at rest.

*Case 4,* N.Z., was a 64 year old white male with arteriosclerotic heart disease. Moist rales were heard over the lower halves of both lung fields, and the patient was considered to be in subacute pulmonary edema. He had received digitalis and Demerol® two hours before the initial study and had been treated with intermittent positive pressure, oxygen and tourniquets. He was studied while in acute pulmonary edema and following recovery two days later.

*Case 5,* F.W., was a 57 year old Negro male with hypertensive cardiovascular disease and uremia. He was producing blood-tinged frothy sputum, and moist rales could be heard bilaterally to the level of the mid-scapulae. He was considered to be in acute pulmonary edema. The patient had not been treated prior to study except for maintenance digitalis. He expired two days after the study and neither autospy nor follow-up study were obtained.

*Case 6,* F.S., was a 54 year old white male with rheumatic heart disease with mitral stenosis and insufficiency. Moist, bubbling rales were heard throughout both lung fields initially, but by the time he was studied these had improved significantly; therefore he was considered in subacute pulmonary edema. Within an hour prior to study he had been treated with aminophyllin, mercuhydrin, digoxin, positive pressure oxygen and tourniquet administration. He was studied in pulmonary edema and following recovery two days later.

*Case 7,* E.B., was a 65 year old white male who had arteriosclerotic heart disease. Moist rales were heard throughout both hemithoraces from the level of the clavicles to the bases, and the patient was considered to be in acute pulmonary edema when first studied. No treatment had been given prior to the initial study. The patient was studied while in acute pulmonary edema and again two days later. He expired suddenly three days after the last study and at necropsy was found to have old and recent myocardial infarction and severe pulmonary edema.

*Case 8,* H.L., was a 58 year old white male with arteriosclerotic heart disease and electrocardiographic evidence of old myocardial infarction. At the time of initial study, coarse bubbling rales were heard diffusely over both lung fields, expiration was prolonged, and wheezes were prominent. He had received morphine approximately three hours prior to study. The patient was studied while in acute pulmonary edema and following recovery three days later.

## REFERENCES


1. Mead, J., Frank, N. R., Lindgren, I., Gaensler, E. A., and Whittenberger, J. L., A technique for the measurement of pulmonary compliance and resistance: its application to normal patients and patients with mitral stenosis. Clin. Research Proc., 1953, 1, 116.
2. Brown, C. C., Jr., Fry, D. L., and Ebert, R. V., The mechanics of pulmonary ventilation in patients with heart disease. Am. J. Med., 1954, 17, 438.
3. Marshall, R., McIlroy, M. B., and Christie, R. V., The work of breathing in mitral stenosis. Clin. Sc., 1954, 13, 137.
4. von Neergaard, K., and Wirz, K., Die Messung der Strömmungswiderstände in den Atemwegen des Menschen, insbesondere bei Asthma und Emphysem. Ztschr. klin. Med., 1927, 105, 51.
5. Frank, N. R., Mead, J., Siebens, A. A., and Storey, C. F., Measurements of pulmonary compliance in seventy healthy young adults. J. Applied Physiol., 1956, 9, 38.
6. Mead, J., Lindgren, I., and Gaensler, E. A., The mechanical properties of the lungs in emphysema. J. Clin. Invest., 1955, 34, 1005.
7. Greene, D. G., Sharp, J. T., Reisman, R. E., and Westinghouse, W. J., Evaluation of pulmonary edema (abstract). Am. J. Physiol., 1955, 183, 622.
8. Moore, J. C., and Sexter, M. S., Changes in lung compliance during development of ANTU pulmonary edema (abstract). Federation Proc., 1956, 15, 132.
9. Cook, C. D., Schreiner, G., and Mead, J., Pulmonary mechanics during induced pulmonary edema in anesthetized dogs (abstract). Federation Proc., 1957, 16, 24.
10. Frank, N. R., Radford, E. P., Jr., and Whittenberger, J. L., Interrelationships of volume-pressure characteristics of lungs and pulmonary blood vessels in excised cat lungs (abstract). Am. J. Physiol., 1955, 183, 617.
11. Borst, H. G., Berglund, E., Whittenberger, J. L., Mead, J., McGregor, M., and Collier, C., The effect of pulmonary vascular pressures on the mechanical properties of the lungs of anesthetized dogs. J. Clin. Invest., 1957, 36, 1708.
12. Mead, J., Whittenberger, J. L., and Radford, E. P., Jr., Surface tension as a factor in pulmonary volume-pressure hysteresis. J. Applied Physiol., 1957, 10, 191.
13. Clements, J. A., Dependence of pressure-volume characteristics of lungs on intrinsic surface-active material (abstract). Am. J. Physiol., 1956, 187, 592.
14. Brown, E. S., Lung area from surface tension effects. Proc. Soc. Exper. Biol. & Med., 1957, 95, 168.
15. Whittenberger, J. L., and Affeldt, J. E., Aspects of pulmonary physiology which determine the effectiveness of artificial respiration procedures. Army Chemical Corps Medical Division, Special Report No. 5, 1951, p. 39.
16. Burton, A. C., Laws of physics and flow in blood vessels *in* Visceral Circulation, Ciba Foundation Symposium. Boston, Little, Brown and Co., 1953. p. 70.