STUDIES ON THE RESPIRATORY MECHANISM IN LOBAR PNEUMONIA.

A Study of Lung Volume in Relation to the Clinical Course of the Disease.

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Plates 35 and 36.

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INTRODUCTION.

The present study has been undertaken in order to obtain data concerning the relationships in pneumonia between the extent and progress of pulmonary involvement, the occurrence of dyspnea and cyanosis, and the changes in lung volume.

In a disease such as lobar pneumonia, it is impossible to measure the vital capacity or the total lung capacity. The patients are too ill to be permitted to make the effort required for maximal inspiration and expiration. Factors, such as psychic state, pleuritic pain, and general muscular weakness would furthermore introduce elements which might easily vitiate the accuracy of measurements involving forced breathing. For this reason it was determined not to attempt to make observations on total lung capacity, vital capacity, or residual air. The mid-capacity (Panum (1); Bohr (2)), we know, forms a fairly constant fraction of the total lung capacity and varies with it. We have chosen the volume of air remaining in the lungs in the resting expiratory position. For this lung volume we have preferred the term given by Lundsgaard (3), functional residual air. This has been studied by Siebeck (4) and Krogh (5) and is sometimes called the mid-capacity. As its name implies, it represents that volume of air which at the end of a normal expiration remains in the lungs and air passages. It must, therefore, express in a quantitative sense, the state of pulmonary distention, and indirectly the surface area of pulmonary epithelium through which diffusion occurs.
Anyone who has made observations on the respiratory movements in man will have noticed that the expiratory pause occurs at a more constant point than the inspiratory. This fact has been emphasized by Krogh (6), Benedict and Collins (7), and Hendry, Carpenter, and Emmes (8). In a graphic tracing of respiration, such as is made in the course of metabolism studies with a Benedict apparatus (Roth (9)), it will be seen that a straight line can be drawn more readily through the expiratory than through the inspiratory points. This has determined us in selecting the expiratory rather than the inspiratory position for measurement.

The functional residual air in a given normal individual is constant within small fluctuations under constant conditions of rest and bodily position. The average deviation for a series of thirteen individuals amounted to but ± 40 cc. in an average lung volume of 2.37 liters. This afforded us our normal criteria. Changes of 100 cc. or more in lung volume measurements are to be regarded as related to the disease process and not as a consequence of normal functional variations or analytical errors.

Methods and Material.

The patient to be investigated was wheeled to the laboratory in his bed. The apparatus was so screened that little of it was exposed to his view. After some preliminary investigation it was found that the ordinary wide flanged rubber mouthpiece was preferable to the mask for pneumonia patients. During the period of dyspnea most patients breathe through their mouths and so do not object to having the nares closed, if it be done gently and gradually. The pressure on the face necessary to make a mask fit snugly produces a sense of suffocation in most patients with pneumonia. We have found it better to have a nurse hold the nose closed than to use a mechanical nose clip. It gives the patient a sense of confidence and is more comfortable. A little vaseline placed in the nares considerably lessens the discomfort of pinching the nose.

Method for Measuring Functional Residual Air.—For measuring functional residual air the method of Van Slyke and Binger (10) was used, with modifications. These consisted of certain technical changes outlined below: (a) a graphic recording device on the mixing spirometer; (b) substitution of all metal parts in the circulation apparatus for the purpose of rendering this more surely proof against leaks and more readily sterilizable; (c) substitution of flutter valves (Sadd valves) in place of Douglas flap valves; (d) substitution of a two-way aluminum stop-cock with minimum dead space for the five-way cock previously used; and (e) collection of gas samples in previously evacuated mercury sampling tubes.
This latter insured almost instantaneous collection of samples from the mixing spirometer at the time desired. Because of the relatively slow mixing of spirometer gas with lung air in normal and shallow breathing, it was necessary to collect the samples at somewhat different intervals than was practised in measuring the

\[
0.95 \times \frac{1}{79.1} \times 2 \text{ (liters of hydrogen)} = 2.40 \text{ liters (uncorrected lung volume)}.
\]

The part of the curve indicated by the broken line is imaginary.

residual air (cf. Van Slyle and Binger (10) and Binger (11)), in which case the first deep inspiration after obtaining vital capacity accomplished most of the gas mixture. The intervals of sampling and the type of mixing curve established from the ratio of a typical case are shown in Text-fig. 1.
A sample was taken at a point, usually within the 1st minute of breathing, before mixture was complete, and three successive samples at \( \frac{1}{2} \) minute intervals were drawn after mixture was known to be complete between the 3rd and 4th minutes of breathing. The \( \frac{N_2}{H_2} \) ratios obtained from these three samples almost invariably fell on a straight line, inclining slightly upward. This upward inclination was discussed by Van Slyke and Binger (10) and can best be interpreted as due to the gradual absorption of \( H_2 \) and elimination of \( N_2 \). It cannot be due to incomplete mixture because a mixture curve could not be straight and would necessarily have diminishing increments. By drawing a curve from the zero point through the point of incomplete mixture to the line representing complete mixture, a small region is delimited in which mixture must be just complete. The mean between points \( A \) and \( B \) (Text-fig. 1) is taken to give the \( \frac{N_2}{H_2} \) ratio from which the lung volume is calculated. In all lung volume determinations in this study we used 2 liters of \( H_2 \) and 2 liters of \( O_2 \) in the mixing spirometer. A correction due to air content in the spirometer amounting to 100 cc. was subtracted from each lung volume determination. This was discussed by Van Slyke and Binger (10). Lung volumes were not reduced to standard temperature and pressure but are given under observed conditions.

A further correction for position was necessary in each determination to assure obtaining the true expiratory position or functional residual air. This was accomplished by the simple procedure of turning the stop-cock, through which the patient
had been breathing room air, while he was in the expiratory phase of respiration. His first breath after connection with the mixing spirometer was therefore into it rather than from it. And the volume of this first breath represented the excess of air in the patient's lungs above that of the resting expiratory position. This is shown in Text-fig. 2. By this procedure the chief source of error in lung volume determinations is obviated.

Text-fig. 3 shows the type of breathing recorded by the mixing spirometer at various stages of the disease in one patient. The slight increase in amplitude at the end of each tracing is due to incomplete scrubbing of air, which permitted slight CO₂ accumulation. The slope in the curves gives an indication of the rate of O₂ consumption. Note the greater respiratory rate and smaller volume of tidal air at the beginning than at the end of disease, and the greater rate of O₂ consumption.

The Instrumental Recording of Respiratory Rate and Depth.—To obtain a quantitative graphic tracing of the pulmonary ventilation in the human subject which will approximate his normal, i.e. unrecorded, breathing is very difficult. Even with every precaution for freedom from mechanical resistance, complete CO₂ removal, and oxygen replenishment, many normal untrained subjects will alter their type of breathing when connected with a spirometer. This alteration is still more pronounced in the pneumonia patient. We have observed the breathing of these patients to be consistently slower, and probably deeper, when it is measured graphically by spirometry than when it is counted at the bedside on the ward. For this reason, in spite of a mass of data on normal subjects and pneumonia patients, we have depended on the counted respiratory rate as evidence of pathological breathing rather than on the measurements made from spirometer tracings. For it is obvious that if the respiratory rate is altered, the minute volume of pulmonary ventilation will likewise be altered. It should be said, however, that in most of the pneumonia patients the characteristic rapid and shallow breathing with increase of minute volume was observed as compared with normal subjects or with the same patient when convalescent. Text-fig. 3 exhibits the progressive changes which are seen in type of breathing during convalescence from the disease. A pause at the end of expiration is usually seen in normal tracings but is missing when breathing is rapid.

The material for study comprised twelve cases of lobar pneumonia of various types. The results from the first two of the series had to be discarded because of errors of technique. Of the remaining ten, seven were men and three women. There was one case of Type I, one of Type II, three of Type III, and five of Type IV. Three of the ten died. The remaining seven recovered without complications. Case 7 had a prolonged period of resolution. Except for the three fatal cases, they were all of rather mild type. For the purposes of the study this was a disadvantage, as few of the surviving cases showed more than slight cyanosis, and in most a fall of temperature followed shortly after admission to the hospital. It would have been of interest to measure lung volumes in that type of case familiar to clinicians in which there is a relatively small area of consolidation but in which
diffuse rales, marked cyanosis, and tachypnea are present; but no such case was available. The lung volumes were never measured on the day of admission, but on the following day when possible, and at succeeding intervals, sometimes daily, sometimes every 2nd or 3rd day, according to the progress of the disease and the change in physical signs. On admission a careful physical and bacteriological examination was made, and on each day of study physical signs and symptoms were

![Text-Fig. 3, a and b. Tracings made by the mixing spirometer at four different periods of disease in Case 7. The first tracing illustrates pronounced rapid and shallow breathing. Time marker indicates 5 second intervals. Signal indicates time at which samples were taken. The increase in amplitude at the end of tracings 1 and 2 is due to slight CO₂ accumulation resulting from insufficient scrubbing](downloaded from May 1, 2017)
noted. X-ray pictures were always taken on the day respiratory tests were made. When there was any marked cyanosis, arterial punctures were done and oxygen analyses made with the Van Slyke constant volume apparatus. An effort was made to express cyanosis in a roughly quantitative way by the use of color scales (Flagg (12); Ridgway (13)). Though these may fairly accurately describe the color of the part, they cannot in pneumonia patients be used with any quantitative accuracy because of the difference in skin color and capillary dilatation in various

of expired air. The upward slope of each tracing represents the rate of $O_2$ absorption, which is notably greatest in tracing 1. The factor for the spirometer is 4.82 cm. excursion of the bell for 1 liter. The scale represents centimeters reduced proportionately to the tracing.
patients and in the same patient from day to day. We have had, therefore, to resort to the unsatisfactory method of describing degrees of cyanosis with plus signs.

**TABLE I.**

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Functional residual air (liters)</th>
<th>Mean (liters)</th>
<th>Average deviation (liters)</th>
<th>Per cent deviation</th>
</tr>
</thead>
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<tr>
<td>D.</td>
<td>M.</td>
<td>3.18</td>
<td>3.20</td>
<td>±0.03</td>
<td>±1.0</td>
</tr>
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<td></td>
<td></td>
<td>3.24</td>
<td></td>
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<td></td>
<td></td>
<td>3.17</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>M.</td>
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<td>3.06</td>
<td>3.04</td>
<td>±0.10</td>
<td>±0.33</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>2.89</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>P.</td>
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<td>2.72</td>
<td>±0.03</td>
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</tr>
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<td></td>
<td></td>
<td>2.68</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Br.</td>
<td>&quot;</td>
<td>2.60</td>
<td>2.62</td>
<td>±0.02</td>
<td>±0.66</td>
</tr>
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<td></td>
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<td></td>
<td></td>
<td>2.65</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.</td>
<td>&quot;</td>
<td>2.53</td>
<td>2.52</td>
<td>±0.01</td>
<td>±0.50</td>
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<td></td>
<td>2.51</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.</td>
<td>&quot;</td>
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<td>2.25</td>
<td>±0.09</td>
<td>±2.25</td>
</tr>
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<td></td>
<td>2.26</td>
<td></td>
<td></td>
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<td></td>
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<td>2.38</td>
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<tr>
<td>T.</td>
<td>&quot;</td>
<td>2.27</td>
<td>2.28</td>
<td>±0.01</td>
<td>±0.50</td>
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<td></td>
<td></td>
<td>2.28</td>
<td></td>
<td></td>
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<tr>
<td>S.</td>
<td>&quot;</td>
<td>1.83</td>
<td>1.89</td>
<td>±0.06</td>
<td>±3.0</td>
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<td></td>
<td></td>
<td>1.95</td>
<td></td>
<td></td>
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</tr>
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<td>C.</td>
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<td>1.77</td>
<td>1.71</td>
<td>±0.04</td>
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<td></td>
<td></td>
<td>1.69</td>
<td></td>
<td></td>
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</tr>
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<td></td>
<td></td>
<td>1.67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>1.53</td>
<td>1.53</td>
<td>±0.00</td>
<td>±0.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.53</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Average: ........................................................................................................... ±1.0
Normal Subjects.

For normal subjects we used physicians and laboratory technicians, all in a good state of health. There were nine men and four women. Functional residual air and pulmonary ventilation were measured with the same technique that was used on the patients. Each subject was required to lie flat on his back in bed for 20 minutes before an observation was made. Physicians are ordinarily very poor subjects for respiratory tests because of consciousness of their breathing during the tests, so that the error in the normal series is probably greater than in the pathological. Furthermore, the normal series was studied first, and with time the accuracy of the technique greatly improved.

Table I shows the average deviation from the mean in repeated determinations of functional residual air in normal subjects. All lung volume determinations in this table were made at rest and with the subjects in the horizontal position.

It follows from the figures given in the table that a variation of ±1 per cent in the lung volume measurements of patients suffering from pneumonia is to be regarded as without significance, since it is within the normal range. We have arbitrarily taken approximately ±5 per cent as a variation assuredly significant of a pathological rather than a physiological change. This is borne out by the observations on the patients.

It was obviously desirable to have some method of predicting the volume of the functional residual air normal to the patients, so that at any time one could tell how far the observed lung volume deviated therefrom. In the cases that recovered, it was easy to establish the patient’s normal by making successive determinations during convalescence and a month or so after discharge from the hospital until constant findings were obtained. An effort was made to correlate the volume of the functional residual air with some physical measurement, such as height, weight, surface area, or chest volume (Lundsgaard and Van Slyke (14)). This was unsuccessful, as is brought out in Table II, in which the cases are arranged in a series according to the volume of the functional residual air, while for each case the other body measurements are given.

The table shows at a glance that functional residual air cannot with any exactness be derived from height, weight, surface area, or chest volume. Take, for example, the two individuals T. and S., who are
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approximately of the same height, weight, and surface area. Notwithstanding this, the volume of the functional residual air in the one case differs from that in the other by about 400 cc. Or take B. and

TABLE II.

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Functional residual air</th>
<th>Height</th>
<th>Weight</th>
<th>Surface area</th>
<th>Chest volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.</td>
<td>M.</td>
<td>3.20</td>
<td>178.6</td>
<td>63.0</td>
<td>1.80</td>
<td>8,660</td>
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<tr>
<td>M.</td>
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<td>3.04</td>
<td>191.3</td>
<td>87.1</td>
<td>2.16</td>
<td>11,640</td>
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<tr>
<td>F.</td>
<td>&quot;</td>
<td>2.72</td>
<td>180.6</td>
<td>77.4</td>
<td>1.92</td>
<td>10,350</td>
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<tr>
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<td>2.62</td>
<td>179.2</td>
<td>66.2</td>
<td>1.84</td>
<td>7,260</td>
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<tr>
<td>L.</td>
<td>&quot;</td>
<td>2.52</td>
<td>182.0</td>
<td>65.5</td>
<td>1.84</td>
<td>8,180</td>
</tr>
<tr>
<td>K.</td>
<td>F.</td>
<td>2.32</td>
<td>170.2</td>
<td>70.0</td>
<td>1.81</td>
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<td>T.</td>
<td>M.</td>
<td>2.28</td>
<td>167.0</td>
<td>65.0</td>
<td>1.73</td>
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<td>180.9</td>
<td>79.5</td>
<td>2.00</td>
<td>10,070</td>
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<tr>
<td>S.</td>
<td>&quot;</td>
<td>1.89</td>
<td>169.0</td>
<td>67.0</td>
<td>1.77</td>
<td>6,550</td>
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<tr>
<td>H.</td>
<td>F.</td>
<td>1.72</td>
<td>159.6</td>
<td>52.1</td>
<td>1.52</td>
<td></td>
</tr>
<tr>
<td>C.</td>
<td>M.</td>
<td>1.71</td>
<td>161.2</td>
<td>58.6</td>
<td>1.62</td>
<td>6,740</td>
</tr>
<tr>
<td>N.</td>
<td>F.</td>
<td>1.53</td>
<td>153.0</td>
<td>60.1</td>
<td>1.57</td>
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</tr>
<tr>
<td>McD.</td>
<td>&quot;</td>
<td>1.37</td>
<td>165.3</td>
<td>58.3</td>
<td>1.65</td>
<td></td>
</tr>
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</table>

T., the volumes of whose functional residual air are within 30 cc. of each other,—less than the experimental error,—and yet they are of quite different shape and size.

The fact that functional residual air could not be definitely correlated with any of these physical measurements suggested that the
level of the diaphragm was the determining factor, and this idea was strengthened by the observation that some of the broad, deep chested, muscular individuals had relatively smaller lung volumes than long chested subjects with relaxed abdominal walls. If the level of the diaphragm determined the volume of the functional residual air, this should shift with a change in body position, and there should be an increase in the functional residual air with alteration from the horizontal to the vertical position, a fact brought out in Table III.

It will be seen that in every instance the functional residual air was greater in the sitting than in the lying position.

Fig. 1 shows an x-ray photograph of Subject P. taken in the lying and in the sitting position. The pictures were exposed as nearly as possible at the end of a normal expiration.

These pictures show that the level of the diaphragm drops after sitting upright and the heart assumes a narrower shape and a more vertical position, which undoubtedly explains the increase in size of lung volume. It seemed obvious from this that no external measurement can be expected to give information about the level of the diaphragm, and the volume of the functional residual air. An attempt to predict the latter from measurements of the area of the lung shadow on the x-ray films, while fairly satisfactory for normal individuals, did not prove so for the pneumonia patients.

Pneumonia Patients.

The changes occurring in the lung volumes of the pneumonia patients are shown in Text-figs. 4 to 13. These charts show the clinical course of the disease as evidenced by the maximum daily temperature, pulse, and respiratory rate. The curve for the average respiratory rate represents the rate for 24 hours on the day of lung volume determination. It will be noticed that, for the reasons stated above, we have not charted the respiratory rate, depth, and minute volume instrumentally recorded, but have preferred to depend upon the respiratory rate counted in the wards. These charts are arranged from a point of view of duration of active disease (fever, rapid pulse, and respirations) after admission to the hospital. Each one is accompanied by a case protocol. The first six cases (Nos. 9, 6, 11, 2, 7, 3; Text-figs. 4 to 9) all show an almost immediate critical fall in tempera-
ture, pulse, and respiration after admission. In Cases 9, 6, 11, and 2 (Text-figs. 4 to 7), the simultaneous increase in lung volume with this critical fall is well demonstrated. In Cases 7 and 3 (Text-figs. 8 and 9), an initial decrease in lung volume was observed (accompanying a spread in physical signs) which occurred in both instances when the patients were still febrile and exhibited accelerated heart rate and rapid breathing, the increase in lung volume not occurring until the more or less critical drop of temperature, pulse, and respirations. The concomitant disappearance of cyanosis should be noted, as well.

In Case 8 (Text-fig. 10), a persistence of fever will be observed from the 3rd to the 8th day after onset. During this period three lung volume determinations were made on the 4th, 6th, and 8th days after onset, respectively. A progressive reduction is seen, accompanied by persistence of cyanosis. This patient had a prolonged period of convalescence with persistent cough and sputum and presence of râles in the right chest. The marked fluctuation in lung volume is shown in Text-fig. 10. In connection with this it should be stated that this case and several others, in which there was marked fluctuation in lung volume, were accompanied in resolution by profuse expectoration, and those in which the lung volume remained constant had relatively scant sputum production.

Cases 4 and 10 (Text-figs. 11 and 12) terminated fatally. In these there was no fall of temperature, pulse, or respiration, and no increase but a decrease in the volume of the functional residual air was observed. Likewise, cyanosis persisted. Case 5 (Text-fig. 13) is presented, in spite of the fact that only one lung volume determination was made, because of the markedly rapid respiratory rate (60 to the minute) and the pronounced oxygen unsaturation of the arterial blood (32 volumes per cent). This case had the smallest volume of functional residual air of any of the series, only 990 cc. in an individual in whom one might anticipate at least 2 to 2.5 liters. The rapid breathing and extreme cyanosis should be noted in association with this marked reduction in lung volume. From an examination of Text-figs. 4 to 13 the conclusion seems justified that functional residual air varies synchronously with the course of the disease as evidenced by temperature, pulse, and respiratory rate. When these fall, the volume of the functional residual air increases. When fever, rapid heart rate,
and rapid respirations persist, the functional residual air either progressively diminishes or remains fixed. Cyanosis similarly in this series of cases disappeared when lung volume began to increase, and was most intense in the three fatal cases in which there were very rapid and shallow breathing and a marked reduction in functional residual air.

It may be questioned at this point whether decrease in functional residual air represents a spread in the pulmonary process and increase is evidence of resolution. We are not prepared to answer this question. The functional residual air represents the volume of air enclosed in the lungs at the end of spontaneous expiration. When this volume is diminished it may be so from a variety of causes other than obliteration of air sacs by consolidation, such as vascular congestion or nervous changes due to modifications in the Hering-Breuer reflex. A close study of the relation of physical signs, x-ray pictures, and functional residual air at different stages of the disease shows them in most instances to vary synchronously and strongly suggests that they all represent different manifestations of the same phenomenon; namely, consolidation and resolution. Fig. 2 illustrates the changes occurring in physical signs, x-ray shadows, and functional residual air observed in Case 8.

Case 9 (Text-Fig. 4).—J. M. Diagnosis: Lobar pneumonia, Type IV. Admitted April 26, 1923, 3 days after onset. Symptoms on admission: Pain in left chest. Headache and fever. Cough with bloody sputum. Physical signs on admission: Dullness over left lower lobe. Coarse moist râles below inferior angle of left scapula. Slight cyanosis of lips, finger-nails, and ears. Some abdominal tenderness. Rusty, blood-streaked sputum.

Clinical Course.—4th day after onset: Moderate cyanosis of lips and finger-nails. Considerable cough with frothy, rusty sputum. Abdominal tenderness in epigastrium. No distention. Dullness over left lower lobe with coarse moist râles.

5th day after onset: No cyanosis. Coughing less severe; copious, frothy, purulent sputum. No abdominal pain. Dullness and coarse moist râles over left lower lobe.

6th day after onset: Occasional cough with scant, frothy sputum. Resonance clearing over left lower lobe; coarse moist râles.

7th day after onset: Slight pain in lower left chest on coughing. Occasional cough with scant, purulent sputum. Impaired resonance with coarse moist râles over left lower lobe; breath sounds clearing.
10th day after onset: Very occasional cough with scant sputum. Resonance only very slightly impaired with distant breath sounds. No râles over left lower lobe.

14th day after onset: No cough or expectoration. Resonance still slightly impaired over left lower lobe; a few fine crackles at the left base.

17th day after onset: No cough. Slightly impaired resonance with distant breath sounds over the left base. No râles.

22nd day after onset: No cough. Resonance clear over left base. Distant breath sounds but no râles heard at left base.
25th day after onset: Discharged. No cough. Still distant breath sounds at left base; normal breathing; no rales. Resolution complete.

46th day after onset: Normal.

Text-Fig. 6. Case 11.
Case 6 (Text-Fig. 5).—J. D. Diagnosis: Lobular pneumonia, Type IV. Admitted April 15, 1923, 5 days after onset. Symptoms on admission: Pain in right chest. Cough and expectoration. Physical signs on admission: Patch of impaired resonance at right base. Moist râles. Dry friction rub below right scapula. Very slight cyanosis of lips and finger-nails. Cough, with frothy, yellowish sputum.

Clinical Course.—6th day after onset: Pain in right posterior chest on breathing. Slight cyanosis of lips and finger-nails. Slight cough with scant sputum. Impaired resonance with moist râles in lower right posterior chest. White blood cells 23,000.

8th day after onset: Slight cough with scant, purulent sputum. Slightly impaired resonance at right base; showers of moist râles. White blood cells 9,375.

10th day after onset: No cough. Very slightly impaired resonance in right paravertebral line with a few moist râles.

13th day after onset: No cough. Very slightly impaired resonance in right paravertebral line with a few fine râles.

22nd day after onset: No cough. Resonance clear at right base. Occasional fine râle in right paravertebral line after forced cough.

64th day after onset: Normal.

Case 11 (Text-Fig. 6).—R. L. Diagnosis: Lobar pneumonia, Type IV. Admitted May 29, 1923, 4 days after onset. Symptoms on admission: Cough. Chilly sensations. Headache. Physical signs on admission: Impaired resonance in upper right chest. Sticky râles to level of fourth rib in right chest. Moderate cyanosis of cheeks, lips, and finger-nails. Cough, with scant, frothy, rusty sputum. Mental confusion.

Clinical Course.—4th and 5th days after onset: Patient quite irrational and actively delirious, requiring restraint. Cough troublesome with scant, frothy, rusty sputum. Moderate cyanosis of cheeks, lips, and finger-tips.

6th day after onset: Loquacious and at times irrational. Considerable unproductive cough. Marked cyanosis of cheeks, lips, and hands after coughing. Consolidation of right upper lobe with moist râles.

7th day after onset: Rational; slight pain in lower right chest. Considerable unproductive cough. Consolidation of right upper lobe with dullness, moist râles, and bronchial breathing.

10th day after onset: Occasional unproductive cough. Showers of coarse, bubbling râles over upper right lobe with increased voice sounds. Resonance only slightly impaired.

12th day after onset: No cough. Resonance clearing over right upper lobe. Scattered fine râles over right apex.

14th day after onset: No cough. Resonance only slightly impaired over upper right lobe. Few fine râles at right apex; voice sounds normal.

17th, 19th, 21st, and 24th days after onset: No cough. Signs over right upper lobe show resolution to be complete. No râles.
Case 2 (Text-Fig. 7).—O. J. Diagnosis: Lobar pneumonia, Type IV; acute fibrinous pleurisy (right). Admitted February 6, 1923, 3rd day after onset. Symptoms on admission: Pain in the right chest. Cough. Blood-streaked sputum. Physical signs on admission: Posteriorly the excursion of the lower half of the right chest is restricted. Resonance impaired below the seventh right rib pos-
teriorly, with bronchial breathing and moist râles. Dry friction rub anteriorly at the level of the right nipple. Slight cyanosis of the lips, finger-tips, ear lobes, and cheeks. Moderate cough with tenacious, rusty expectoration.

Clinical Course.—4th day after onset: Slight pain in the anterior right chest on coughing. Cough less severe, expectoration rusty and less copious. Impaired resonance below the sixth rib of the right posterior chest with coarse râles and bronchial breathing. Perspiration profuse. Slight cyanosis of the lips, finger-nails, ears, and cheeks. White blood cells 28,125. Arterial oxygen: content 18.9 volumes per cent; capacity 20.7 volumes per cent; arterial unsaturation 10 per cent.
6th day after onset: Moderate cough with scant, brown, purulent sputum. Moderate perspiration. No cyanosis. Slightly impaired resonance with scattered râles in the right paravertebral line about the midscapular region. A dry friction rub is still heard about the right nipple area anteriorly.

11th day after onset: No cough or expectoration. A few scattered moist râles are heard along the right paravertebral line.

34th day after onset: Patient has been discharged for 9 days and returns for lung volume determination. He has no complaints and is gaining weight and strength. Physical examination shows apparently a normal right lung.

Case 7 (Text-Fig. 8).—M. W. Diagnosis: Lobar pneumonia, Type II; acute pleurisy. Admitted April 17, 1923, 4 days after onset. Symptoms on admission: Pain in left chest. Shortness of breath. Cough. Physical signs on admission: Dullness and râles below inferior angle of left scapula. Dry pleural rub in left axilla. Slight cyanosis of lips, cheeks, and finger-nails. Cough, with rusty, bloody sputum.

Clinical Course.—5th day after onset: Pain in left chest on coughing or breathing. Definite cyanosis of lips, cheeks, and finger-nails. Slight cough with scant, rusty sputum. Slight abdominal distention. Consolidation of left lower lobe below the inferior angle of the left scapula. Moist râles heard above lower left lobe, and a few dry râles at the right base. White blood cells 10,625.

6th day after onset: Slight cyanosis of lips and finger-nails. Coughing very little, unproductive. No abdominal distention. Signs of consolidation with râles below inferior angle of left scapula. Oxygen content of arterial blood 17.5 volumes per cent; capacity 19.5 volumes per cent; unsaturation 10 per cent.

8th day after onset: No cyanosis. Moderate cough with scant, purulent sputum. Impaired resonance over left lower lobe; coarse, bubbling, moist râles over left lower lobe.

10th day after onset: Very slight unproductive cough. Slightly impaired resonance over left lower lobe, breath sounds more normal, with moist râles.

12th day after onset: Occasional unproductive cough. Resonance only slightly impaired over the left lower lobe; scattered fine crackles at left base.

20th day after onset: Resonance clear at left base. No râles. No cough.

27th and 63rd days after onset: Normal.

Case 3 (Text-Fig. 9).—W. G. Diagnosis: Lobar pneumonia, Type I. Admitted March 23, 1923, 4 days after onset. Symptoms on admission: Headache. Pain in left chest. Cough. Physical signs on admission: Dullness in lower left chest. Râles below the fourth rib left posterior chest. Slight cyanosis of lips and finger-nails. Cough, with rusty, blood-tinged sputum.

Clinical Course.—4th day after onset: Following intracutaneous horse serum test patient developed marked anaphylactic shock symptoms with dyspnea, cyanosis, accelerated pulse, cough, widespread urticaria, nausea, vomiting, and diarrhea.
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7th day after onset: Slight cyanosis of lips and finger-nails. Frequent cough, with scant, purulent, blood-streaked sputum. Consolidation of left lower lobe with a few moist râles. White blood cells 12,812.

8th day after onset: Frequent cough with small amount of frothy, rusty sputum. Resonance clearing over left lower lobe; very few moist râles heard.

10th day after onset: Slight cough with very scant, purulent sputum. Consolidation still present in left lower lobe with very few moist râles. Slight pain in left chest.

12th day after onset: Respiratory rate increased. Occasional cough with scant expectoration.

14th day after onset: Occasional unproductive cough. Resonance impaired over left lower lobe. Scattered coarse, moist râles over left lower lobe.

18th day after onset: Slight cough, no expectoration. Impaired resonance over left lower lobe with scattered fine râles.

22nd day after onset: No cough or expectoration. Very slight impaired resonance over left lower lobe, few fine râles, normal breath sounds.

31st day after onset: No cough. Resonance clear, no râles. Resolution complete.

36th day after onset: Normal.

Case 8 (Text-Fig. 10).—C.M. Diagnosis: Lobar pneumonia, Type III. Admitted April 25, 1923, 3 days after onset. Symptoms on admission. Pain in right shoulder. Cough with blood-streaked sputum. Headache. Physical signs on admission: Dullness and moist râles and increased voice sounds over upper right lobe. Few moist râles at left base. Considerable cough with small amount of tenacious, rusty, bloody sputum. Slight cyanosis of lips, ears, and finger-nails.


6th day after onset: Shortness of breath. Pain in right chest on coughing. Moderate cyanosis of lips and finger-nails. Cough less severe; sputum bloody and rusty. Moderate abdominal distention. Dullness and moist râles, apparently spread to upper part of middle and lower right lobes. White blood cells 21,875.

8th day after onset: Crisis during the early morning followed by profuse perspiration. Considerable coughing with tenacious, rusty, blood-streaked sputum. Resonance clearing in right upper lobe with coarse bubbling râles and dullness of middle and right lower lobes with bronchial breathing.

10th day after onset: Moderate cough with yellowish, purulent sputum. Resonance clearing from above downward to the right base with widespread, coarse bubbling râles throughout.
11th day after onset: Feeling of tightness in right side of chest on taking a deep breath. Moderate cough with tenacious, frothy, purulent sputum. Resonance clearing to third rib anteriorly and seventh rib posteriorly, with coarse bubbling râles, while below are dullness and bronchial breathing.

12th day after onset: Moderate cough with yellowish, purulent sputum. Resonance clearing over right chest; bronchial breathing disappeared. Coarse bubbling râles throughout.

13th day after onset: Moderate cough with scant, tenacious, purulent sputum. Resonance slightly impaired over posterior right chest; widespread moist râles with a few dry râles heard throughout right chest.

14th day after onset: Pain on pressure over the third and fourth ribs anteriorly and on taking a deep breath. Coughing moderately with tenacious, slightly rusty, and blood-streaked sputum today. Physical signs same as on last day.

16th day after onset: Occasional cough with tenacious, purulent, slightly blood-streaked sputum. Slightly impaired resonance over the right chest; breath sounds faint; showers of fine moist râles over right chest.

17th day after onset: Occasional cough with few tenacious globules of purulent sputum. Signs unchanged.

23rd day after onset: Occasional cough with a few globules of purulent, yellowish sputum. Slightly impaired resonance with showers of fine râles in the right axilla.

27th day after onset: Occasional cough with scant purulent sputum. Showers of fine râles with distant breath sounds in right axilla.

30th day after onset: Still occasional cough with small amount of sputum. Slightly impaired resonance with showers of fine râles in right axilla and radiating posteriorly and anteriorly.

34th day after onset: Very occasional unproductive cough. Numerous fine moist râles with slightly impaired resonance in right axilla.

40th day after onset: Slight unproductive cough. Fine moist râles persist in the right axilla and over the lateral border of middle right lobe.

45th day after onset: Occasional unproductive cough. A very occasional fine moist râle is heard in the right axilla after a deep breath.

Case 4 (Text-Fig. 11).—K. O. Diagnosis: Lobar pneumonia, Type III. Admitted March 13, 1923, 2 days after onset. Symptoms on admission: Cough, Fever, Coryza. Physical signs on admission: Impaired resonance below the seventh rib, right posterior, with suppressed breath sounds and moist râles. Respirations rapid and shallow. Slight unproductive cough.

Clinical Course.—3rd day after onset: Troublesome cough with scant amount of tenacious, rusty, bloody sputum. No cyanosis. Dullness, bronchial breathing, and increased voice sounds over right lower lobe. A few râles heard at left base posteriorly. White blood cells 21,875.

4th day after onset: No cyanosis. Moderate cough with scant, rusty sputum. Dullness and definite consolidation of whole right lower lobe. A few râles heard at the left base. White blood cells 23,125. Blood from hand vein: oxygen content
13.95 volumes per cent; oxygen capacity 16.35 volumes per cent; oxygen unsaturation 15 per cent.


*Blood was drawn from the hand vein. This has been shown recently (Goldschmidt, S., personal communication) to agree closely in its oxygen content with arterial blood.

**Text-Fig. 11.** Case 4.

*Case 10 (Text-Fig. 12)._J. McG. Diagnosis: Lobar pneumonia, Type III, with Pneumococcus Type III bacteremia. Admitted May 15, 1923, 3 days after onset. Symptoms on admission: Cough with blood-streaked expectoration. Pain in left chest. Fever. Physical signs on admission: Restricted movement of
left lower chest with dullness, moist râles, and bronchial breathing over the left lower lobe. Slight cyanosis of lips, cheeks, and finger-nails. Cough with rusty, bloody sputum.


4th day after onset: Respiration rapid and shallow, mostly abdominal in character. Moderate cyanosis of cheeks, lips, ears, and hands. Moderate cough with frothy, brown sputum. Consolidation of whole left chest with palpable rhonchi over both lungs. Few râles at right base. White blood cells 21,250.
5th day after onset: Pain in nape of neck and lower lumbar region. Respirations rapid and shallow, mostly abdominal. Marked cyanosis of cheeks, lips, ears, and hands. Moderate cough with frothy, brownish sputum. Consolidation of left lung complete, with dullness and rales. A patch of dullness and rales at right base posteriorly. White blood cells 26,250. Blood from hand vein: oxygen content 13 volumes per cent; capacity 18.1 volumes per cent; unsaturation 28 per cent. Death.

Case 5 (Text-Fig. 13).—E. M. Diagnosis: Lobar pneumonia, Type IV. Admitted March 24, 1923, 4 days after onset. Symptoms on admission: Pain in left chest. Cough with blood-tinged sputum. Fever. Physical signs on admission: Dullness over left lower lobe. Moist rales over left lower lobe. Breathing rapid, thoracoabdominal in type. Moderate abdominal distention. Cough with scant, rusty, blood-streaked sputum.
Clinical Course.—6th day after onset: Pain in left chest on coughing and breathing. Cyanosis of lips, cheeks, ears, and finger-nails. Frequent unproductive cough. Moderate abdominal distention. Consolidation with dullness and râles over whole left chest. Right chest shows areas of dullness and diffuse moist râles. White blood cells 5,625. Arterial blood: oxygen content 10.6 volumes per cent; oxygen capacity 15.6 volumes per cent; oxygen unsaturation 32 per cent. Death.

Addendum.—Since the preparation of this article Levy (15) has published a paper showing that the size of the heart in pneumonia increases during the course of the disease and returns to a constant gradually. Comparison of his figures1 with ours is of interest in respect to the cardiac changes which apparently must occur simultaneously with those in lung volume. If one were to superimpose our diagrams on his one would be led to believe that cardiac dilatation occurred at the same time that lung volume was reduced.

SUMMARY AND CONCLUSIONS.

1. The functional residual air (defined as the lung volume at the end of normal expiration) has been determined in a series of normal individuals and in ten patients with lobar pneumonia at different stages of the disease.

2. The rate, depth, and minute volume of respirations were measured in the same individuals by a graphic method.

3. When appreciable cyanosis was present the oxygen content and capacity of the arterial blood were determined.

4. A constant relationship has been found to exist between the persistence and disappearance of symptoms (fever, accelerated heart rate, rapid and shallow breathing, cyanosis) and fluctuations of the functional residual air. When these symptoms persisted the functional residual air decreased; during their disappearance the volume of the functional residual air rose towards normal. The rise was detected soon after the crisis.

5. A close parallelism has been observed also between alterations in radiographic shadow, physical signs, and the volume of the functional residual air. The lung volume, measured at normal expiration, is diminished during the persistence of pathological signs in the lungs, and returns to normal as the pathological signs disappear. The average time required, in cases which recovered, for the functional residual air to become constant was 11 to 12 days, counting from the onset of the disease.

1 Levy (15), Figs. 5, 7, and 9.
BIBLIOGRAPHY.


EXPLANATION OF PLATES 35 AND 36.

Fig. 1. Radiographic pictures of a normal subject (P.) showing changes in position of diaphragm and heart accompanying alteration in the subject's position from lying to sitting. The functional residual air appropriate for the position is indicated.

Fig. 2, a to e. Diagrams indicating coincident changes in radiographic shadow, physical signs, and functional residual air in a patient at different intervals during the process of consolidation and resolution. The lines indicate impaired resonance; dots indicate the presence of râles.
Normal Subject P.

Lying 2.71 liters. Sitting 4.05 liters.

Fig. 1.

4th day after onset.

1.68 liters functional residual air.

Fig. 2, a.

6th day after onset.

1.54 liters functional residual air.

Fig. 2, b.

(Bläger and Brow: Respiratory mechanism in pneumonia.)
8th day after onset.

1.41 liters functional residual air.  

14th day after onset.  

Fig. 2, c.  

1.57 liters functional residual air.  

40th day after onset.  

Fig. 2, d.  

1.66 liters functional residual air.  

Fig. 2, e.  

(Binger and Brow: Respiratory mechanism in pneumonia.)