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VITAL CAPACITY OF THE LUNGS: CHANGES OCCURRING IN HEALTH AND DISEASE

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In 1854 Andrews (1), of the University of Michigan, wrote of the spirometer ". . . I am inclined to adopt the opinion of others who maintain that this instrument cannot be relied upon for diagnosis except where the previous vital capacity of the individual is known, so as to decide whether there is really a diminution going on. At the same time it must be confessed that diseases of the lungs exert a remarkable influence on the capacity." One year before this, Fabius (2), in his inaugural dissertation on the vital capacity of the lungs, had also suggested making determinations during health in order that, should sickness supervene, the former might serve as a basis of comparison with that secured after the onset of disease. However, these suggestions have largely gone unheeded, most of the publications from the time of Hutchinson (3) to the present being concerned with the variations from standards based upon body measurements rather than with actual changes in the vital capacity, incident to disease.

In 1924, with the publication with Kornblum (4) of an article on the vital capacity, the writer decided to employ the suggestions of Fabius and Andrews, and see whether the value of the vital capacity as a diagnostic procedure would be enhanced thereby. It was decided that an attempt should be made to secure the vital capacity of every patient presenting himself for office treatment, regardless of the nature of his complaint, and that, in addition, the vital capacity of certain available groups of healthy individuals should be studied.

Vital capacity changes in youthful healthy subjects

The first group to be considered consisted of 482 healthy students in Drexel Institute, Philadelphia. (See Table I.) Half of them were men ranging in age from 17 to 30 years with an av-

erage age of 21.36 years, the other half were women, aged 16 to 25, the average age being 20.14 years.

TABLE I
Ages of normal subjects

Age years	Men	Women
16	0	1
17	1	4
18	23	24
19	47	68
20	53	82
21	43	37
22	42	18
23	19	5
24	7	1
25	3	1
26	2	0
27	0	0
28	0	0
29	0	0
30	1	0
Total	241	241
Mean age	21.36 years	20.14 years

In this group the vital capacity figures of one year were compared with those of the next. It will be seen (Fig. 1) that many variations occurred both above and below the readings of the previous year; that both men and women exhibited an average gain, and that the men gained more than the women. The gain of the men exceeds three times the standard error of the mean and is statistically significant, that of the women is not.

It was interesting to compare these findings with the variations from the hypothetical vital capacity as calculated from the standards of West (5) which are based upon surface area. As would be expected, the variations from West's standard (Fig. 2) were considerably greater than those from the reading of the previous year, showing that the latter offered a better basis for predicting the vital capacity than did West's surface area standard.

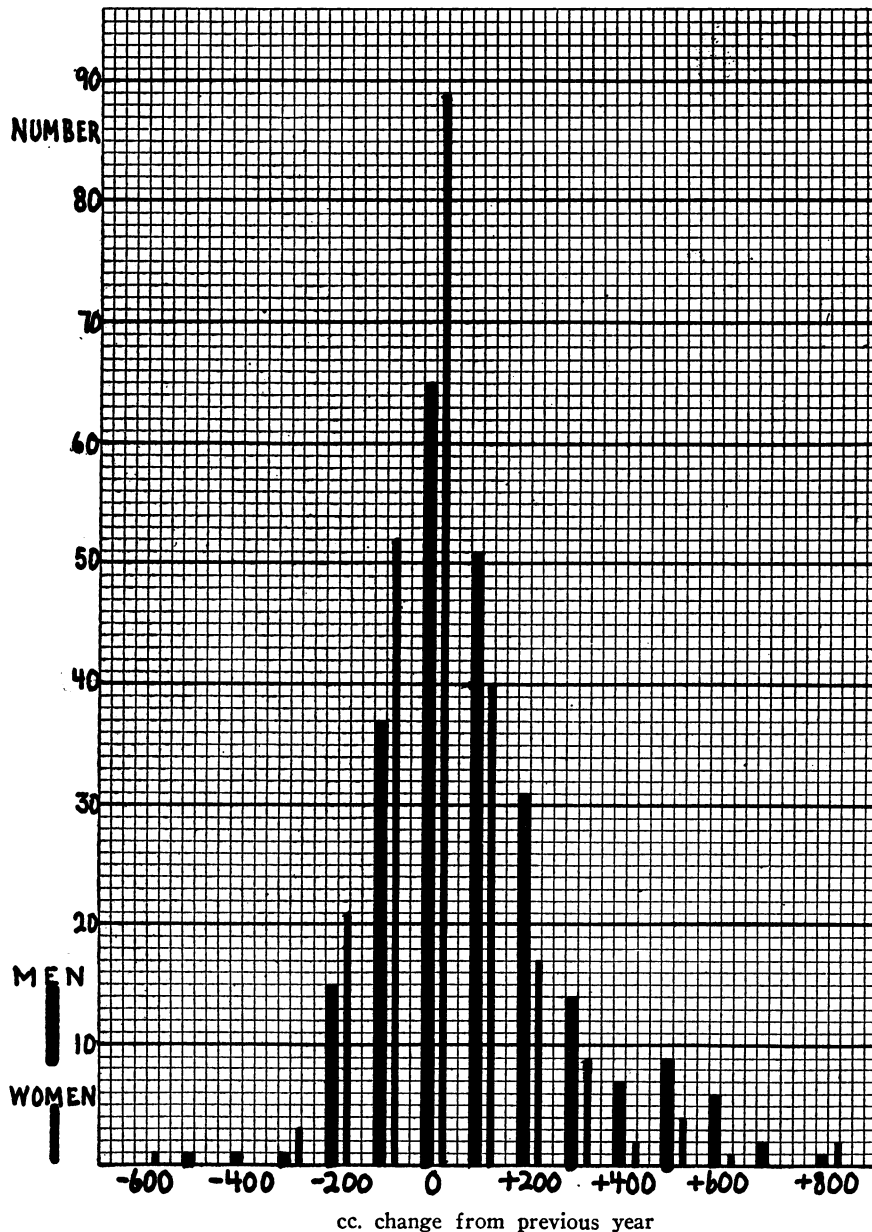


FIG. 1. VITAL CAPACITY CHANGES FROM PREVIOUS YEAR IN 482 NORMAL SUBJECTS.

Standard deviation = 202 cc. for men, 169 cc. for women. Standard error of the mean = 13 cc. for men, 11 cc. for women. Mean gain in vital capacity = 89 cc. for men, 17.4 cc. for women.

The data, furthermore, help to answer the question: "How far below the normal figure can the vital capacity fall before the diminution should be regarded as significant?" Adopting three times the standard deviation as the criterion of significance in the case of the student whose vital capacity is compared with that of the previous

year, a diminution exceeding 695 cc. for the men and 507 cc. for the women would be regarded as significant.¹ On the other hand, where a student's

¹ For the men, the standard deviation (202 cc.) \times 3 = 606 cc. Adding the mean gain (89 cc.) gives 695 cc. For the women, the standard deviation (169 cc.) \times 3

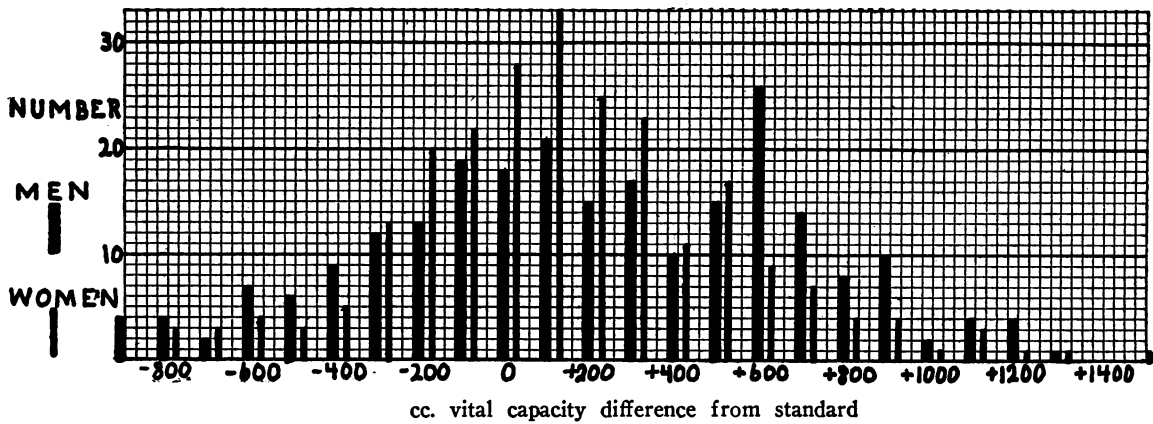


FIG. 2. VITAL CAPACITY VARIATIONS FROM WEST'S STANDARD IN 482 NORMAL SUBJECTS.

Standard deviation = 482 cc. for the men, 385 cc. for the women. Standard error of the mean = 31 cc. for the men, 25 cc. for the women. Mean difference from West's standard = + 198 cc. for the men, + 137 cc. for the women.

vital capacity is compared with the figure calculated by West's formula, readings more than 1,644 cc. below West's standard in the case of men, and 1,292 cc. below in the case of women may be considered significant.²

Vital capacity changes in acute bronchitis

Having determined what variations were to be expected in the vital capacity of youthful subjects in health, a group of youthful patients with acute bronchitis was next studied. A difference of opinion regarding the effects of bronchitis upon the vital capacity is manifested in the literature, some writers believing that little if any diminution is produced, while others hold that a marked lowering of the vital capacity is to be expected. Table II shows these patients divided into ambulatory and bed cases. As might be expected the bed cases exhibited a greater average loss than the ambulatory ones. From a statistical standpoint the loss in vital capacity in the ambulatory group could not be regarded as significant; in the bed cases, however, the difference is almost 3 times

gives 507 cc. (the gain is not significant and is therefore not added).

² For the men, the standard deviation (482 cc.) × 3 gives 1,446 cc. Adding the mean difference from West's standard (+ 198 cc.) gives 1,644 cc. For the women, the standard deviation (385 cc.) × 3 gives 1,155 cc. Adding the mean difference from West's standard (+ 137 cc.) gives 1,292 cc.

TABLE II

Vital capacity changes in acute bronchitis
(Cases grouped as ambulatory or bed)

Vital capacity difference from healthy figure cc.	Number of ambulatory patients	Number of bed patients
+ 400	1	0
+ 300	1	0
+ 200	4	2
+ 100	11	4
0	13	3
- 100	9	6
- 200	9	5
- 300	0	2
- 400	1	1
- 500	1	0
- 600	0	1
Total	50	24
Standard error of the mean	23.2 cc.	38 cc.
Standard error of the mean × 3	69.6 cc.	114 cc.
Mean loss in vital capacity	20 cc.	100 cc.

the standard error of the mean, and probably is significant. However this may be, a study of Table II indicates that in most instances the vital capacity would have afforded little assistance in differentiating bronchitis from normality. On the other hand, this very fact indicates that the vital capacity might be of assistance in differentiating that disease from others which regularly cause a significant diminution in the vital capacity, pneumonia, for example.

The vital capacity changes in pneumonia

Although it has long been known that the vital capacity is greatly diminished in pneumonia, yet,

except in postoperative pneumonia (6) vital capacity determinations prior to and during the onset of this disease have heretofore been lacking. In Cases 1 and 2 it was possible to secure fairly complete sets of vital capacity readings, before, during, and after pneumonia. (See Figs. 3 and 4.)

the physical signs were by no means characteristic of pneumonia, there being skodaic resonance over the right base, little if any alteration in the breath sounds and few râles over the right lower lobe. The patient never looked acutely ill, and she improved daily so that on the 14th day of the disease a roentgenogram showed the chest to be entirely negative, and she was discharged. Twelve days later she returned to her normal activities.

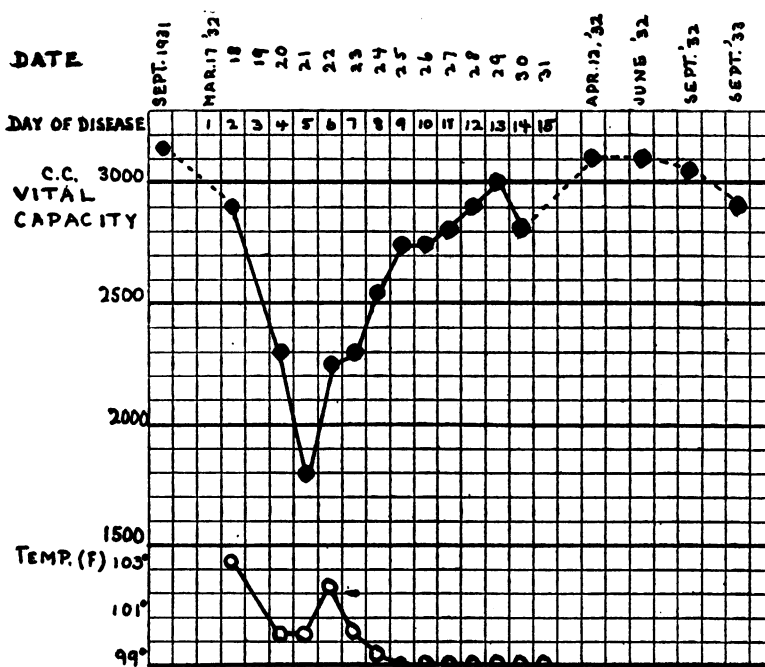


FIG. 3. VITAL CAPACITY BEFORE, DURING AND AFTER MILD INFLUENZAL PNEUMONIA (CASE 1, FIG. 5A).

A loss of 250 cc. by the second day of the disease is shown, then a further loss of 1,100 cc. followed by a rapid rise approximately to the figure secured prior to the illness. Here, and in Figure 4, the highest temperature of each 24 hours is indicated by a circle.

Case 1. Miss V. B., aged 20, suffered from unreduced congenital bilateral dislocations of both hips: otherwise her past medical history was negative. Her vital capacity in health was found to be 3,100 cc. (See Fig. 3.) Six months later she was seized with pain in the abdomen and back, and on the following day she developed a chill, cough and fever of 102.8° F. The chest examination was negative. On the next day her leukocyte count was 8,500 and râles appeared at the base of the right lung. Because of the fall in her vital capacity, and despite the atypical findings, pneumonia was diagnosed and she was sent to the University Hospital. On the 6th day of her disease her leukocytes numbered 19,600 and a roentgenogram showed "density at the right base, the appearance of which is that of pneumonic consolidation." (See Figs. 3 and 5A.) Even at this time

Case 2. Miss M. S., aged 20, in health had a vital capacity of 3,500 cc. Six months later she was seized with a sore throat and a fever of 103° F. On the following day she had a leukocytosis of 11,400 and examination of the chest revealed the classical signs of pneumonia over the lower lobe of the right lung. She was taken to the University Hospital where she ran a course characteristic of lobar pneumonia, the upper as well as the lower lobe becoming involved by the pneumonic process (see Figs. 4 and 5B). On the 33d day of the disease she was discharged from the hospital, and 29 days later resumed her normal activities.

These cases illustrate the diminution in vital capacity which regularly accompanies pneumonia. In Case 1 the fall was gradual and the disease

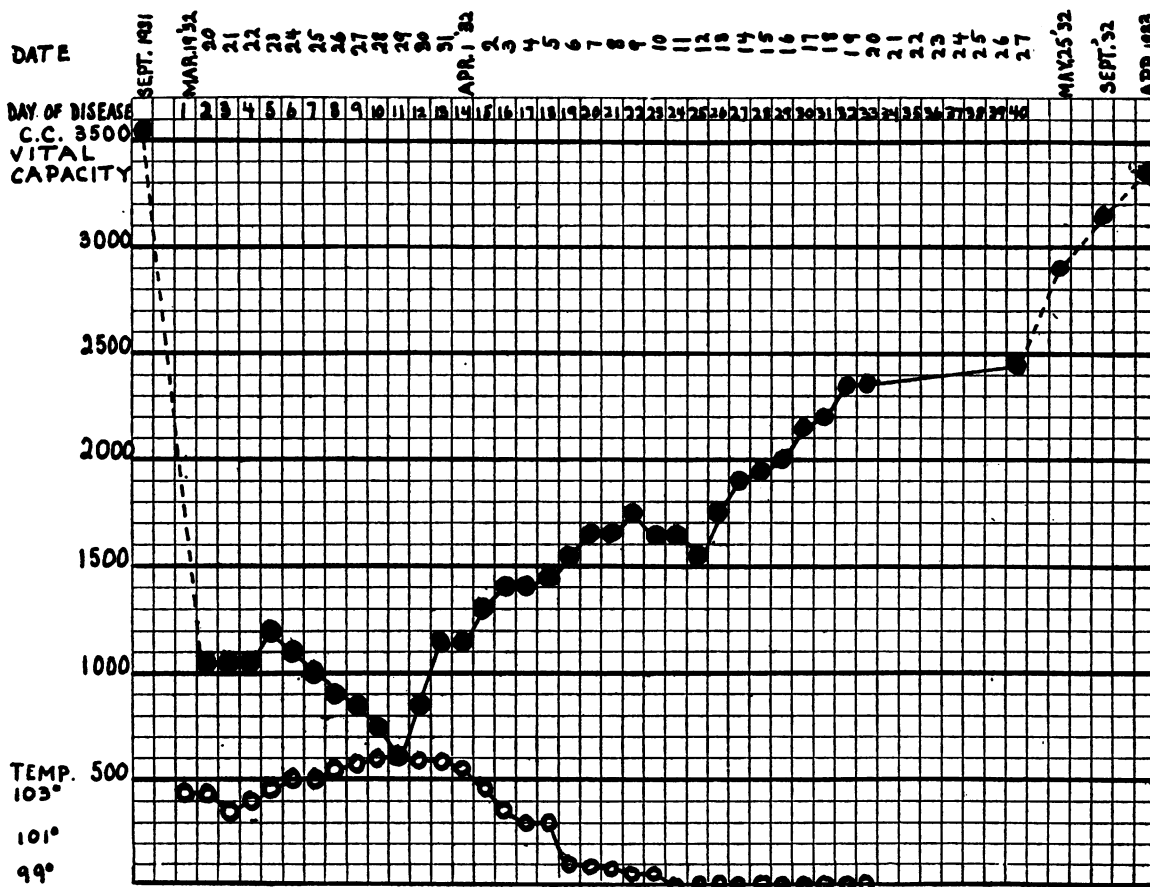


FIG. 4. VITAL CAPACITY BEFORE, DURING AND AFTER LOBAR PNEUMONIA INVOLVING FIRST THE RIGHT LOWER, THEN THE RIGHT UPPER LOBE OF CASE 2 (FIG. 5B).

A loss of 2,500 cc. by the second day of the disease is shown, then a further loss of 450 cc., followed by an increase beginning before subsidence of the temperature has begun. The vital capacity 1 year and 7 months after the attack is shown to be very little below that secured prior to its onset.

mild. Here the vital capacity was of distinct service, strongly suggesting the diagnosis of pneumonia. It is possible that cases of mild pneumonia masquerading as bronchitis may be more frequent than is generally recognized, and if so, the vital capacity should assist in their recognition. Case 2 exhibited a more precipitous and profound diminution in the vital capacity than did Case 1. The physical findings and course of the disease were so characteristic of lobar pneumonia that neither the vital capacity nor roentgenogram were needed to make the diagnosis. It is interesting to note how early in the disease the fall in the vital capacity occurred and how an increasing vital capacity was the first evidence of beginning lysis. Both in this patient and in Case 1 there

was eventually a return of the vital capacity to approximately the pre-pneumonia level. That such does not always occur is evidenced by the following case.

Case 3. A man, aged 40, measuring 70½ inches and weighing 145 pounds, whose vital capacity in health was 6,000, developed a prolonged attack of streptococcal bronchopneumonia which resulted in the diaphragmatic adhesion shown in Figure 5C. Although in perfect health during the subsequent 5 years the vital capacity has remained 5,500 cc.

In spite of the loss of 500 cc. his vital capacity is still 1,000 cc. above the normal figure as predicted on the basis of West's standard. This case illustrates how a complication such as pleuritic adhesions may lead to a permanent diminution in the

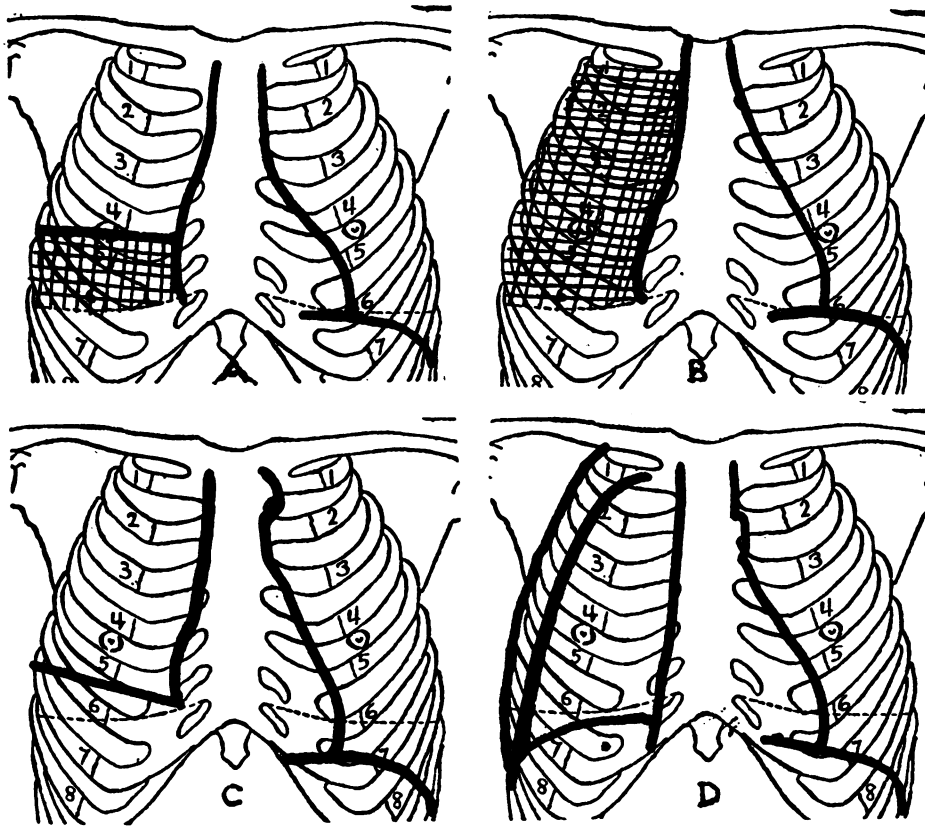


FIG. 5. ROENTGENOGRAPHIC FINDINGS.

A. Case 1. Mild atypical pneumonia involving the right lower lobe. The vital capacity loss was 1,350 cc. (see Fig. 3).

B. Case 2. Right upper and lower lobe pneumonia. The vital capacity loss was 2,950 cc. (see Fig. 4).

C. Case 3. Adherent right diaphragm due to preceding bronchopneumonia resulting in a permanent loss of 500 cc. in vital capacity.

D. Case 4. Spontaneous pneumothorax of right lung. The vital capacity loss was 500 cc.

vital capacity and how relying upon West's standard one might conclude that the vital capacity was above normal whereas a loss of 500 cc. is known to have occurred.

Vital capacity changes in spontaneous pneumothorax

The literature contains a number of observations upon patients under treatment with artificial pneumothorax in whom vital capacity determinations were made both before and after the introduction of air (7). The following case, however, is apparently the first in which the vital capacity before and after the occurrence of a spontaneous pneumothorax is reported.

Case 4. Mr. C. S., aged 20, consulted the writer on March 21st, 1928, because of pain over the right eye which appeared following swimming. The positive physical findings were: blood pressure 140/85, left tonsil red, right frontal sinus dark on transillumination. His vital capacity at this time was 4,300 cc. Two weeks later he presented himself perspiring, pale and complaining of pain in the right chest which was aggravated by breathing or coughing. The onset was sudden and followed moderately violent exertion. The breath sounds over the right chest were slightly diminished but other chest findings, including the coin test, were negative. The vital capacity was found to be 3,800 cc., a loss of 500 cc. from that of 2 weeks before. The roentgenogram (see Fig. 5D) revealed partial collapse of the right lung due to pneumothorax. One month later the vital capacity had returned to within 100 cc. of the original figure, the

chest examination was negative, and the roentgenogram showed complete expansion of the affected lung.

Vital capacity in heart disease

Although it has been recognized since the time of Kentisch (8) that congestive heart failure caused marked diminution of the vital capacity, yet, as far as the writer is aware, Case 5 is the first in which the vital capacity before, during and after the first attack of decompensation is reported.

Case 5. Mr. W. H., aged 57, consulted the writer in August, 1928, because of pharyngitis. His vital capacity was 3,400 cc. (900 cc. below the figure predicted from West's surface area standard.) He gave a history of having had a retinal hemorrhage 6 years before and of hypertension for an indefinite period, but he had never suffered from cardiac decompensation. He was not heard from again until December 22d of the same year, at which time he suffered from cough and dyspnea on exertion, his vital capacity being 2,900 cc. His heart, which previously had shown little or no enlargement, now extended well outside the mamillary line and a systolic murmur had become audible over the apex. An electrocardiogram made at this time revealed inverted T waves in Lead I, diphasic T waves in Lead II and notched QRS waves in Lead III. The cough grew worse and on January 11th, 1929, he was orthopneic, the vital capacity being 1,800 cc. This represented a loss of 1,100 cc. in 20 days and a total loss of 1,600 cc. since August. Under treatment in the hospital he gradually improved and on February 11, 1931, cardiac compensation was well established and his vital capacity was 3,500 cc.

This case is of interest in showing the gradual diminution of the vital capacity incident to decompensation and the return of the vital capacity to its original level as compensation becomes restored.

SUMMARY

1. The vital capacity in two consecutive years was determined in a group of 482 youthful

healthy subjects. Deviations from the readings of the previous year proved to be decidedly smaller than those from the hypothetical vital capacity as calculated from the surface area formula of West. The reading of the previous year, therefore, constituted the more reliable basis for vital capacity prediction.

2. In a group of 74 cases of acute bronchitis an average diminution from the previously determined healthy figure was noted in both ambulatory and bed cases, a more marked diminution occurring in the latter.

3. The vital capacity is shown before, during and after illness, in cases of pneumonia, spontaneous pneumothorax and cardiac decompensation. These observations are believed to be unique.

BIBLIOGRAPHY

1. Andrews, E., Vital capacity. *Peninsular J. Med.*, 1854, 1, 530.
2. Fabius, H., *De Spirometro Ejusque Usu*. Inaugural dissertation. Amsterdam, 1853.
3. Hutchinson, J., On the capacity of the lungs and on the respiratory functions, with a view of establishing a precise and easy method of detecting disease by the spirometer. *Med. Chir. Trans.*, 1846, 29, 137.
4. Arnett, J. H., and Kornblum, K., Vital capacity; an inquiry into its value as a diagnostic procedure. *Ann. Clin. Med.*, 1924, 3, 255.
5. West, H. F., Clinical studies on the respiration. VI. A comparison of various standards for the normal vital capacity of the lungs. *Arch. Int. Med.*, 1920, 25, 306.
6. Powers, J. H., Vital capacity. Its significance in relation to postoperative pulmonary complications. *Arch. Surg.*, 1928, 17, 304.
7. Myers, J. A., and Bailey, W., Studies on the respiratory organs in health and disease. XX. The value of the vital capacity test in artificial pneumothorax treatment. *Am. Rev. Tuberc.*, 1925, 10, 597.
8. Kentisch, E., *An account of baths*. London, 1814.