Archives of Internal Medicine

VOL. 28

NOVEMBER, 1921

No. 5

CLINICAL STUDIES OF THE RESPIRATION. VII

THE EFFECT OF GENERAL WEAKNESS AND FATIGUE ON THE VITAL CAPACITY OF THE LUNGS*

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The clinical significance of spirometry in disease was recognized more than sixty years ago, but in the last decade little attention has been devoted to its practical application. Recently, however, a number of studies on the respiration have again indicated that the character and possible extent of the pulmonary ventilation are intimately related to the vital capacity of the lungs, and that determination of the vital capacity gives important information as to the functional efficiency of the lungs. Clinical observations have shown that the vital capacity of the lungs is of especial interest in pulmonary tuberculosis¹ and in heart disease,² and that in both these conditions it serves as an index of the clinical condition of the patient. As the disease becomes more severe, the vital capacity falls, and as the disease improves, the vital capacity increases and approaches the normal.

In pulmonary tuberculosis and in the other inflammatory or infiltrative lesions of the lung substance, as well as in conditions which, like pleural effusion, interfere with the movements of the lungs, it is quite easy to understand why the vital capacity is reduced. In heart disease the circumstances are often less simple. The tendency to dyspnea on exertion increases progressively as the vital capacity falls, and there is abundant evidence to indicate that the interference with pulmonary ventilation is an important factor in producing dyspnea, but, particularly in the early stages of heart disease, an increased tendency to dyspnea associated with a decrease in vital capacity may be present without any physical signs of involvement of the lungs or pleural

*From the Medical Clinic of the Peter Bent Brigham Hospital.

1. Dryer, G., and Burrell, L. S. T.: *Lancet* 1, 1212, 1920.

Wittich, F. W., Myers, J. A., and Jennings, F. L.: *J. A. M. A.*, **75**:1249 (Nov. 6) 1920.

2. Peabody, F. W., and Wentworth, J. A.: *Arch. Int. Med.* **20**:443 (Sept.) 1917. McClure, C. W., and Peabody, F. W.: *J. A. M. A.* **69**:1954 (Dec. 8) 1917.

cavities. A similar condition may also prevail in late stages, for in many cases the extent of the physical signs is not at all in proportion to the great fall in the vital capacity of the lungs. Thus a cardiac patient with moderate pleural effusion and atelectasis may have much lower vital capacity than a patient with a tuberculous pleural effusion of the same extent. It has been suggested that the limitation of the movements of the lungs in heart disease, of which the low vital capacity is an indication, may be due to changes in the pulmonary circulation, but proof of this is as yet lacking, and the question naturally arises as to whether the low vital capacity may not be due to general muscular weakness. Does the low vital capacity in heart disease depend on the fact that cardiac patients are feeble and easily fatigued? If the diminution in the vital capacity is due to general weakness, then its value as a clinical test is distinctly limited, for it merely gives an indication of how feeble or exhausted the patient is. If, on the other hand, the fall in vital capacity depends largely on local changes and on the severity or extent of the specific disease, then the test has a much greater clinical significance, for it provides one more method by which the development and retrogression of the disease may be followed. The question has an important practical bearing, and it has, therefore, seemed worth while to attempt to determine how great a factor general muscular weakness may be in causing a reduction of the vital capacity of the lungs. This problem has been approached by us in two ways. In the first place, the vital capacity of the lungs has been determined in a group of patients in whom weakness and a tendency to fatigue was very great, but in whom there was no evidence of disease of the heart or lungs. In the second place, patients with heart disease and a low vital capacity have been studied and an attempt made to fatigue the muscles of respiration.

1. THE EFFECT OF GENERAL WEAKNESS ON THE VITAL CAPACITY OF THE LUNGS

It has proved to be much more difficult than was anticipated to find suitable cases in which to determine the effect of general weakness on the vital capacity of the lungs. This is chiefly due to the fact that all patients with pathologic conditions of the heart or lungs had to be excluded as these lesions in themselves cause a marked decrease in the vital capacity. The patients selected were almost all suffering from pernicious anemia, and they are particularly well adapted to the purpose because the outstanding symptoms of the disease are general weakness and a tendency to dyspnea on exertion, while the involvement of lungs and circulation is usually slight. One case of typhoid fever is included, the first observation being made on the second afebrile day after a severe course of thirty-five days of fever, at a time when

the patient was extremely weak, and the second observation five weeks later when she had been getting up in a chair for nine days. It was, of course, necessary to choose patients who were willing to cooperate satisfactorily, and with one or two exceptions they are all below 50 years of age. This selection by age was imperative for it is well known that there is normally a decrease in the vital capacity of the lungs after the age of 50. The normal standards on which the percentage of vital capacity is based are those of West,³ according to which the vital capacity is equal to 2.5 liters per square meter surface area in young men, and 2.0 liters per square meter surface in young women. The surface area was determined by the chart of DuBois and DuBois⁴ and depends on height and weight. The observations are shown in Table 1.

2. ATTEMPT TO FATIGUE THE MUSCLES OF RESPIRATION

If the low vital capacity of the lungs in heart disease is due wholly, or in large part, to general weakness or to weakness of the muscles of respiration, it would be natural to expect a fatigue effect to show itself if strain is put on the respiratory muscles. In these observations, the attempt was made to produce such a fatigue effect by taking the vital capacity every 15 seconds for 10 minutes. The method is similar to that employed by Levine and Wilson⁵ in studying patients with "irritable heart." The exertion involved in the test is considerable, and it would not have been surprising to find a gradual decrease in the volume of the maximum expiration as the experiment progressed. This is what was to be expected if weakness and fatigue play an important part in the production of the low vital capacity. As will be seen, no such tendency for the volume of expiration to decrease was observed. Five patients were studied. Four had serious cardiac lesions and vital capacities between 32 and 55 per cent. of the normal standards. One patient had pernicious anemia and he was so weak that he could hardly stand on his feet long enough to have his height measured. His vital capacity was 79 per cent of normal. The highest of the first three observations is taken as the vital capacity in each case. The following protocols show the results of these tests of vital capacity.

PROTOCOLS OF OBSERVATIONS

CASE 1.—W. J. R., male aged 38 years; height, 165 cm.; weight, 68.4 kg.; surface area, 1.76 sq. m.

Diagnosis.—Aortic stenosis and insufficiency; mitral stenosis.

Physical Examination.—Patient is sitting propped up in bed and looks dyspneic. Lungs are negative, except for impairment of percussion note at the right base posteriorly below the angle of the scapula, with slight suppression

3. West, H. F.: Arch. Int. Med. **25**:306 (March) 1920.

4. DuBois, D. and DuBois, E. F.: Arch. Int. Med. **15**:868 (June) 1915.

5. Levine, S. A., and Wilson, F. H.: Heart **7**:53, 1919.

TABLE 1.—DETERMINATION OF VITAL CAPACITY

No.	Name	Sex*	Med. No.	Age	Height in Cm.	Weight in Kg.	Surface Area in Sq. M.	Hb. %	Erythrocytes	Leukocytes	Vital Capacity in C.c.	Per Cent. of Normal Vital Capacity	Diagnosis	Remarks
1	V. A.	♀	5485	21	160.5	49.0	1.50	67	2,736,000	4,400	3,500	116	Pernicious anemia	Two months history of weakness
2	M. J. A.	♀	3718	36	137.0	43.0	1.39	35	1,400,000	4,900	2,730	98	Pernicious anemia	Duration of present illness 4 years. Chief complaint "weakness."
3	P. H. B.	♂	4223	46	169.0	72.0	1.83	25	980,000	1,600	3,650	80	Pernicious anemia	Marked weakness for 2 years
4	M. C.	♂	4181	51	160.0	52.0	1.52	43	2,170,000	5,600	3,470	91	Pernicious anemia	Chief complaints "extreme weakness and vomiting."
5	T. J. H.	♂	3729	17	163.0	44.3	1.45	36	1,192,000	5,850	3,050	84	Anemia, secondary, due to gasoline fumes	Quit work three weeks before admission on account of weakness and dizziness
6	E. A. L.	♀	4316	48	162.0	48.3	1.50	50	1,490,000	4,200	2,685	90	Pernicious anemia	Onset of present illness one year ago with gradually increasing weakness
7	F. A. M.	♀	4565	38	161.0	46.0	1.46	72	2,520,000	8,000	2,725	94	Pernicious anemia	Four months history of weakness and diarrhea
8	H. M. S.	♀	4478	38	169.0	62.4	1.72	75	3,728,000	10,200	2,550	74	Pernicious anemia	Unable to work for past three months on account of weakness
9	J. T. S.	♂	3863	43	168.0	75.6	1.87	61	2,440,000	4,800	4,550	97	Pernicious anemia	Duration of present illness five months. Quit work three weeks ago on account of weakness
10	J. S. D.	♂	14764	40	168.0	63.4	1.72	35	1,072,000	2,300	3,300	77	Pernicious anemia	Could hardly stand on account of weakness
11	A. H.	♂	15262	42	175.0	51.0	1.60	45	1,400,000	1,200	3,150	79	Pernicious anemia	Duration of present illness twenty months; history of weakness, diarrhea; not able to do even light work for one month
12	R. C.	♀	15018	46	151.0	43.2	1.34				2,300	86	Typhoid fever	Test made on thirty-sixth day of typhoid fever, second day of normal temperature. Could not register grip testing apparatus with either hand
13	A. P. T.	♂	15642	47	171.0	53.6	1.62	35	1,824,000	4,800	4,200	104	Pernicious anemia	Weakness and ease of fatigue chief complaints

* In this column, ♂ denotes male, and ♀ female.

of breath sounds and diminished vocal fremitus over this area. A few scattered râles are present at both bases behind. There is edema of the lower extremities.

Vital Capacity: 1,400 c.c., or 32 per cent. of normal.

Vital capacity test every 15 seconds:

1300, 1400, 1200, 1300, 1350, 1350, 1450, 1400, 1400, 1400,
1400, 1400, 1400, 1350, 1400, 1400, 1475, 1475, 1500, 1450,
1475, 1450, 1400, 1450, 1400, 1500, 1475, 1500, 1450, 1575,
1550, 1500, 1500, 1500, 1500, 1550, 1550, 1550, 1550, 1500.

CASE 2.—A. J. C., male, aged 50 years; height, 175 cm.; weight, 73 kg.; surface area, 1.88 sq. m.

Diagnosis.—Chronic myocarditis. Patient is comfortable while lying in bed but gets short of breath walking on level or upstairs.

Physical Examination.—Heart is much enlarged and there is a systolic murmur at the apex. There are a few râles at the bases of both lungs, and slight dullness with diminished intensity of breath sounds at extreme base on right.

Vital Capacity: 2,400 c.c., or 51 per cent. of normal. (Six months ago, while he was still able to work, his vital capacity was 3,600 c.c.)

Vital capacity tests every 15 seconds:

2350, 2400, 2300, 2100, 2300, 2600, 2700, 2650, 2700, 2600,
2650, 2550, 2400, 2450, 2400, 2450, 2550, 2400, 2375, 2650,
2700, 2700, 2100, 2500, 2450, 2650, 2300, 2600, 2500, 2600,
2500, 2350, 2400, 2600, 2450, 2600, 2400, 2600, 2600, 2450.

CASE 3.—A. G., female, aged 54 years; height, 152 cm.; weight, 80.4 kg.; surface area, 1.77 sq. m.

Diagnosis.—Chronic myocarditis. The patient entered the hospital with cardiac decompensation and auricular fibrillation. She is now much improved and the heart is regular. She has just begun to get out of bed.

Physical Examination.—Heart is much enlarged, and there is a systolic murmur at the apex. Examination of the lungs is absolutely negative.

Vital Capacity: 1,500 c.c., or 42 per cent. of normal.

Vital capacity tests every 15 seconds:

1200, 1500, 1350, 1450, 1250, 1500, 1350, 1300, 1600, 1400,
1500, 1600, 1300, 1550, 1600, 1300, 1475, 1600, 1700, 1650,
1750, 1600, 1400, 1475, 1600, 1650, 1550, 1800, 1300, 1800,
1700, 1800, 1700, 1600, 1600, 1300, 1750, 1600, 1650, 1900.

The subject tried very hard and always made a prolonged expiration. This made the intervals between tests very short. Twice she was bothered by cough and the intervals after the fifth and thirty-second tests were 30 seconds.

CASE 4.—L. D., female, aged 58 years; height, 159 cm.; weight, 72 kg.; surface area, 1.73 sq. m.

Diagnosis.—Chronic myocarditis. Patient entered the hospital badly decompensated and has improved under digitalis. She is still in bed constantly.

Physical Examination.—Heart is much enlarged, with a systolic murmur at the apex. Lungs are entirely negative, except for scattered râles at both bases posteriorly.

Vital Capacity: 1,900 c.c., or 55 per cent. of normal.

Vital capacity tests every 15 seconds.

1900, 1900, 1800, 1850, 1700, 1700, 1800, 1900, 1450, 1850,
1900, 1800, 1900, 1900, 1900, 2000, 1850, 1900, 1950, 1850,
1850, 1880, 1800, 1800, 1900, 1889, 1820, 1840, 1800, 1900,
1900, 1800, 1900, 1850, 1820, 1800, 1600, 1800, 1700, 2050.

Expiration was always much prolonged and on two occasions the intervals amounted to 30 seconds.

CASE 5.—J. S. D., male, aged 40 years; height, 168 cm.; weight, 63.4 kg.; surface area, 1.7 sq. m.

Diagnosis.—Pernicious anemia. Patient has been sick a long time and was so weak that he could hardly stand to be measured. Hemoglobin, 35 per cent.; erythrocytes, 1,073,000; leukocytes, 2,300.

Physical Examination.—Heart and lungs are negative.

Vital Capacity: 3,300 c.c., or 78 per cent. of normal.

Vital capacity tests every 15 seconds:

3150, 3300, 3300, 3200, 3200, 3250, 3200, 3200, 3100, 3200,
3100, 3150, 3200, 3100, 3000, 3200, 3300, 3200, 3300, 3400,
3300, 3400, 3450, 3300, 3400, 3450, 3300, 3300, 3400, 3400,
3450, 3400, 3400, 3300, 3350, 3400, 3300, 3400, 3400, 3400.

An attempt was made to obtain some quantitative conception of the actual strength of patients by means of testing the strength of the grip of the hands and to correlate this with the vital capacity. The method is, of course, very unsatisfactory but the relative results as given in Table 2 are of interest. They indicate that there is no relation between muscular strength as tested by this method and the vital capacity of the lungs.

TABLE 2.—MEASUREMENTS OF STRENGTH OF PATIENTS

Patient	Sex*	Grip		Vital Capacity, C.c.	Vital Capacity, Per Cent. of Normal	Diagnosis
		Right	Left			
A. J. C.	♂	91	81	2,400	51	Cardiac
W. J. R.	♂	80	78	1,100	32	Cardiac
A. G.	♂	30	25	1,500	42	Cardiac
L. D.	♂	25	20	1,900	55	Cardiac
A. H.	♂	60	50	3,150	79	Pernicious anemia
J. S. D.†	♂	66	45	3,300	78	Pernicious anemia
R. C.‡	♀	0	0	2,300	86	Typhoid fever

* In this column, ♂ indicates male, and ♀ female.

† It is, perhaps, noteworthy that J. S. D., the strength of whose grip was fairly good, was so weak that he could hardly stand.

‡ The patient had such extreme weakness that she was unable to register even the slightest change on the machine used to test the grip.

DISCUSSION

Observations on the vital capacity of the lungs in patients with extreme general weakness but without definite disease of the heart or lungs and without other local cause which would affect the pulmonary ventilation showed surprisingly little variation from the normal. In the series of cases reported, the vital capacity was in no instance less than 74 per cent. of the normal standards based on body surface area. In connection with this, it must be remembered that most normal persons have a vital capacity of at least 90 per cent. of the normal standards, but that in a small number (about 5 per cent.) of healthy young adults the vital capacity has been found to be between 85 and 90 per cent. of the normal. On this basis then, one may say that extreme general weakness may account for a decrease in the vital capacity of the lungs

of, roughly, from 20 to 30 per cent. below the normal standards. The number of patients examined is small, however, and exceptional cases may show a somewhat greater decrease in vital capacity due to weakness. As patients with severe heart disease may have a vital capacity that is 75 per cent. below the normal² it is obvious that general weakness is not a significant factor in accounting for this decrease. Further evidence against the suggestion that fatigue and general weakness play an important rôle in reducing the vital capacity of the lungs in heart disease is the fact that in patients with a low vital capacity the taking of many observations of the vital capacity at short intervals does not result in a falling off of the volume of the respiration as might occur in fatigue.

Another possible factor which might be considered as explaining the inability to breathe deeply in heart disease would be an abnormality of the Hering-Breuer reflex so that the stimulus to expiration or inspiration came prematurely and before the other respiratory phase was complete. Against this hypothesis is the fact that even with severe cardiac disease it is usually possible for patients to hold the breath a considerable time at the end of either full inspiration or expiration. The impulse to take up the other phase of respiration is not extremely pressing. Indeed, the deep expiration is often very prolonged. Most patients with heart disease are willing to cooperate completely in the attempt to determine the vital capacity of the lungs. The result, therefore, as anyone who has had experience will admit, represents the "real" vital capacity and the low value is not due to the fact that the patient is "not trying hard." It is usually simple to pick out the patient who will not cooperate and give what is actually his maximum expiration, and in such case the results must, of course, be discarded as worthless. The evidence here presented supports the conception that the decrease in the vital capacity of the lungs in heart disease is due to a local cause, and is not the result of general weakness. This is of importance for the cause of the decrease, while not as yet wholly clear, is probably intimately associated with the cardiac condition. If the low vital capacity depends on the condition of the heart and circulation and not merely on general weakness, then the determination of the vital capacity has a greater specific significance than would otherwise be the case.

A discussion of the effect of general physical condition on the vital capacity of the lungs would be incomplete without reference to the recent observations made on the vital capacity in cases of "irritable heart" or "effort syndrome." It is characteristic for patients classed in these categories to have physical weakness with a marked tendency to fatigue and breathlessness on exertion combined with a strongly

neurasthenic temperament. The most complete study on the vital capacity in this condition is that of Levine and Wilson⁵ who examined 131 British soldiers, grouping them according to the class in which they were discharged from the hospital. When compared with the normal standards of Peabody and Wentworth² the average vital capacity for each class was as follows: Class 1: 97 per cent.; Class 2: 92 per cent.; Class 3: 90 per cent.; "permanently unfit": 87 per cent. The only class, therefore, in which the vital capacity fell below the generally accepted normal standards was the last, and, considering the poor general condition of men discharged from the army as "permanently unfit," it is surprising that the vital capacity was not lower. This evidence supports the view that general physical condition is not a factor which causes large variations from the normal in the vital capacity. The work of Adams and Sturgis,⁶ who studied the vital capacity in 100 American soldiers with "irritable heart," shows similar results, for only seven men had a vital capacity of less than 85 per cent. of the normal. Tests of muscular strength showed no relation between the muscular development and the vital capacity. Observations of great interest were also made by White⁷ on patients with "effort syndrome" and on neurotic patients with "shell shock." The average vital capacity of his normals was 4,720 c.c.; of five patients with "effort syndrome," 3,250 c.c.; and of five patients with "shell shock," 2,240 c.c. The same relative inefficiency of the "effort syndrome" and neurotic groups was found in applying other respiratory tests such as holding the breath, measuring the expiratory force by means of a manometer, and the "blow-bottle" test. White concludes that the vital capacity proved to be rather a test "of nervous stability than of the condition of the cardiovascular or respiratory systems per se, in the groups under discussion." There can be no question as to the correctness of this conclusion, and of the certainty that these groups of men would fail in any test which involved concentration, persistence, or vigorous action. The asthenic state is characteristic of the soldier with "shell shock" or "effort syndrome" but the underlying factor is weakness of will and not weakness of muscle. The same thing is, of course, true of the neurasthenic of civil life. Where the will is weak the vital capacity of the lungs may apparently be low, but the observation will have no value as indicating disease of the heart or lungs because it does not represent a true value. Such patients will not exert themselves enough to give what is actually their maximum respiration. Levine and Wilson's experience in testing the effect of fatigue on the

6. Adams, F. D., and Sturgis, C. C.: *Am. J. M. Sc.* **158**:816, 1919.

7. White, P. D.: *Am. J. M. Sc.* **159**:866, 1920.

vital capacity in patients with "irritable heart" is interesting in this connection. They found that immediately after exercise the vital capacity in normal subjects was essentially the same as before exercise, while in soldiers with "effort syndrome" there was a decrease, which in the most severe cases amounted to 18 per cent. Likewise, in the patients with severe "effort syndrome" if vital capacity determinations were made in rapid succession so that 30 readings were made in 10 minutes there was a progressive fall in the volume of the expirations which in some instances amounted to 25 to 50 per cent. This is in direct contrast to our patient with pernicious anemia (No. 5) who was so weak that he could hardly stand but who was able to keep up the volume of his vital capacity when forty observations were made in 10 minutes. The striking feature was that in spite of great general muscular weakness the respiratory muscles seemed to maintain a fairly good strength. The will of this patient was strong, his cooperation was good and he was willing to exert himself to his utmost so that the situation was quite different from that with patients with "effort syndrome." Muscular weakness, therefore, has comparatively little effect on the vital capacity of the lungs, but weakness of the will or failure to cooperate and give the maximum expiration are factors which render observations on the vital capacity worthless and misleading. Fortunately, these difficulties are not encountered very frequently in patients with organic disease, and are usually easily recognized when present.

In contrast to the effects of muscular weakness on the vital capacity it is of interest to recall some facts with regard to the effect of physical training. Among eighty-five normal young men whom West³ studied in his attempt to establish normal standards for vital capacity there were twenty-five who gave a history of an unusual amount of athletics and physical exercise. Only two of these had a vital capacity below the average normal standard; in ten the vital capacity was not more than 10 per cent. above normal; in eight it was between 10 and 20 per cent. above normal; and in five it was between 20 and 30 per cent. above normal. The general range of increase of vital capacity above the normal standards associated with the best physical training is thus approximately the same in amount as the decrease below the standards due to extreme physical weakness.

CONCLUSIONS

1. The vital capacity of the lungs in patients with great physical weakness, but without disease of the heart or lungs, was found to be not more than 26 per cent. below the normal standards. In heart disease, the vital capacity may be as much as 75 per cent. below the normal standards.

2. Repeated tests of the vital capacity were made every 15 seconds for 10 minutes in patients with severe heart disease. The exertion involved was considerable, but no evidence of fatigue of the muscles of respiration was observed. The volume of the maximum expiration was as great at the end of the series of tests as at the beginning.

3. These observations indicate that general muscular weakness and fatigue of the muscles of respiration are not important factors in causing the reduction of the vital capacity of the lungs in heart disease.