THE VITAL CAPACITY DETERMINATION

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The vital capacity of the lungs is the volume of air which can be expired after the deepest possible inspiration.

Hutchinson, in 1846, invented a spirometer and pointed out its extreme value in the diagnosis of early pulmonary tuberculosis. Until recently, however, the value of the vital capacity determinations was not appreciated, and it is safe to predict that it will not be long before the vital capacity will be an indispensable part of the complete physical examination.

Before the vital capacity determination can be of any practical value, it is essential to have:

1—a method and technic which is simple and accurate, and at the same time which is not time consuming,

2—a normal standard for comparison,

3—a knowledge of the various factors which may influence the vital capacity.

METHOD

A spirometer built according to the specifications described by Peabody permits great accuracy and is best adapted for use with children and adults. The adjustment of a self-recording dial is of great advantage, since the entire attention may be given to the proper performance of the test. The subjects stand or sit upright in bed or on a chair, and breathe in and out as deeply as possible through a rubber or glass mouthpiece, the nose being closed by a tight clip. No corrections need be made for temperature, pressure or water tension.

It is not difficult to obtain satisfactory determinations of the vital capacity in adults, and even children respond with surprising intelligence and ease. Care is taken always to explain to the patients the desired object, and it is necessary to urge them to breathe as deeply as possible. Strict attention should be paid to this point, for otherwise the volume of the respiration will be low and will not represent the true maximum of respiratory ex-

change. The first attempt is often unsatisfactory, but the second or third attempt will invariably give reliable results. The observations are made rapidly, and consume from three to five minutes for each patient.

VITAL CAPACITY OF NORMAL ADULTS

The approximate normal vital capacity of an individual must be known before readings are of much significance in the diagnosis or prognosis of disease. The normal vital capacity for men and women of average physical fitness has been computed by Dreyer and others who used the height, weight, chest measurements, stem length or surface area as the basis for their calculations. The normal vital capacity varies considerably with such factors as obesity, age, occupation and previous physical training and experience. An athletic person or one who plays a wind instrument or takes other strenuous exercise will have a greater vital capacity than an inactive person. All these factors must be taken into consideration. A vital capacity below 15% of the normal may be looked upon as being of pathologic significance.

The recent work tends to confirm the original observation made by Hutchinson of the relationship between the vital capacity and height, and of its tremendous variability, although it is more constant than compared with weight. Lungsgard and Van Slyke contend that chests measured according to certain formulas and found to agree in size would more nearly have the same vital capacity than would persons of the same height, a contention which is denied by Peabody. Christie and Beams, using the "linear formula" of DuBois, have demonstrated that the surface area and not the height or weight or chest measurements is by far the most constant and exact standard for comparison. This confirms the observations made by Dreyer and by West. From observations on 290 normal men and women, Christie and Beams conclude that "a female from 20 to 30 years of age, with a body surface of 1.4 to 1.5 square meters, has a vital capacity of 2,700 c. c., and for each gain of 0.1 square meter in body surface the vital capacity goes up about 175 c. c. A male from 20 to 30 years of age, with a body surface of from 1.6 to 1.7 square meters, has

a vital capacity of about 4,000 c. c., and with each gain of 0.1 square meter the vital capacity goes up 350 c. c. From these data, we can conclude that a normal male between the ages of 20 and 30 has a vital capacity of 2.5 liters per square meter of body surface, and that a normal female between 20 and 30 has a vital capacity of 2 liters per square meter of body surface.'' The maximum respiratory exchange remains practically constant up to 50 years, after which there is a gradual decline—greatest between 50 and 60 years, and reaching 50 per cent of the normal at 85 years.

RESPIRATORY DISEASES

I. *Tuberculosis.*

Meyers, studying the vital capacity in tuberculosis, found a direct relation between the extent of pulmonary involvement and the degree of the lowered vital capacity.

On the basis of roentgen-ray examination, he classed the cases as follows:

I. Suspected cases in which roentgen-ray examination was negative.
II. Tuberculous cases.
    A. Peribronchial with unilateral and bilateral involvement.
    B. Parenchymatous
        1. Unilateral with
            a—the disease confined above the first rib or an area of similar size.
            b—With the disease extending below the first rib but not involving more than one lobe.
            c—involving more than one lobe.
        2. Bilateral.
            a—Disease confined above the first rib, or an area of similar size on each side.
            b—Disease extending below first rib, on each side but not involving more than two lobes.
            c—Involvement of more than two lobes.

In thirty suspected cases in which the roentgenograms showed no evidence of disease, the mean vital capacity was 102 per cent of the normal, the range being 82 per cent to 122 per cent of the normal.

In thirty-nine cases showing unilateral peribronchial tuberculosis, the average vital capacity was 97% and the range from 81 to 121% of the normal.

Seventy-one cases of unilateral and seventy with bilateral parenchymatous tuberculosis revealed by the stereo roentgenograms were grouped according to the extent of involvement. The vital capacity of the groups was found to decrease as the extent of the disease increased, the average being 74%, and the range from 26 to 122% of the normal.

On the basis of physical examination, in ninety cases showing no evidence of tuberculosis or in which a definite diagnosis was not made, 90% gave vital capacities within normal limits, while variations of 70% to 90% occurred in the remaining 10%.

In 172 cases showing varying degrees of pulmonary disease, the vital capacity was found to be decreased in proportion to the extent of involvement. The vital capacities of 30 cases, showing pulmonary cavities by the roentgen-ray, ranged from 31 to 109% of the normal, the average being 64%. Nine cases of spontaneous pneumothorax showed vital capacities averaging 49%, the range being 32% to 58% of the normal.

III. *Bronchial Asthma.*

Peabody, Wentworth and Barker, and others have reported vital capacity readings in patients suffering from bronchial asthma, and found that in some cases the lung volume was considerably decreased, and in others, it was normal.

Meyers, reporting 20 cases in which the readings were taken at various times during and between the attacks, found the vital capacity was reduced tremendously during the attacks, in some cases to 20% of the normal, which quickly returned to the normal as the attack disappeared. In four cases in which the disease had extended over a period of years, the vital capacity did not return to nor-

mal limits after the disappearance of the asthmatic attacks. In these cases, physical and roentgen-ray examinations revealed definite evidence of emphysema, which apparently was sufficient to account for the reduced lung capacity.

IV. *Pneumonia.*

In pneumonia, Meyers found the vital capacity to be very low from the beginning, being reduced to 50% or less in most cases. This marked reduction from the beginning of a case of pneumonia, he points out, is almost diagnostic. The lowest vital capacity was observed on or near the day of crisis. From this time on, if the patient recovers, the vital capacity gradually increases through convalescence. An increase in the vital capacity is noted almost immediately after the crisis, but does not return to normal for many days. The amount of pulmonary consolidation bears no relationship to the vital capacity, as is true also of the relationship between consolidation and dyspnea, as pointed out by Means and Barach. They also point out that the dyspnea may increase after the crisis, with no evidence of alteration in the anatomic processes in the lungs. This, however, does not hold true for the vital capacity, as an increase is noted almost immediately following the crisis. The test, therefore, is a valuable aid in the early diagnosis of pneumonia. In a case of unresolved pneumonia, the vital capacity remained almost stationary for a period of ten days following the crisis. In another case, in which the tubercle bacilli appeared in the sputum, the vital capacity remained stationary over a long period of time.

Peabody and Wentworth suggested that the reduced vital capacity present in patients convalescing from pneumonia may occur in any severe acute infection. Peabody and Sturgis studied the effect of fatigue and general weakness on the vital capacity, using patients suffering from pernicious anemia. In none of these cases, without a lung or heart disease, was the vital capacity reduced more than 26% below normal. In another series of cases, attempts were made to fatigue the muscles of respiration by taking the vital capacity every fifteen seconds for ten minutes. Contrary to what might be expected,

the vital capacity was as great at the conclusion of the experiment as at the beginning. The investigators conclude 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."

V. *Pleural Effusion.*

In a group of cases which include hydrothorax, pneumothorax, hemothorax, and empyema, the vital capacity was found to vary between 74% and 42% of the normal. The vital capacity seems to depend upon the amount of fluid or air in the pleural cavity, and there is a close relationship between the tendency to dyspnea and the decrease in the vital capacity.

DISEASES OUTSIDE RESPIRATORY TRACT

I. *Nephritis.*

In eight cases of acute nephritis with no history of dyspnea, the vital capacity was within normal limits. In chronic nephritis, without evidence of heart disease, and without a history of dyspnea, the vital capacity was high, and within normal limits. In cardiorenal cases, dyspnea was a prominent symptom, and the vital capacity usually was decreased in proportion to the intensity of the dyspnea.

II. *Hyper-Thyroidism.*

Dyspnea on exertion is a common symptom complained of by patients with Graves' disease. This may be due to nervousness, but usually indicates cardiac weakness. The decrease in the vital capacity corresponds to the tendency to dyspnea.

III. *Paratyphoid Fever.*

Meyers, studying the vital capacity in acute diseases outside the respiratory tract, found only 15% of the cases in an epidemic of paratyphoid fever, with vital capacities below normal. In more than half of these cases, the reduced vital capacity could be explained on the basis of complications, such as pleurisy or lung involvement.

From these observations, we may conclude that the vital capacity may be of value in suggesting pulmonary complications in diseases outside the respiratory tract.

VITAL CAPACITY OF THE LUNGS IN HEART DISEASE

It has long been known that the vital capacity of the lungs is frequently decreased in heart disease. Peabody and Wentworth confirmed this fact by making 224 observations on 124 patients, and showed in a striking manner that the clinical condition of the patient, and more especially the tendency to dyspnea, varied directly with the degree of diminution of the vital capacity. They subdivide the cases into four groups, basing their classification on the degree of diminished vital capacity.

Group One consists of cases with a vital capacity of 90 per cent or more of the normal. Very few of these patients complained of any symptoms referable to their hearts. Many of them entered the hospital for other diseases, and the cardiac condition was discovered in the course of the routine examination. The vast majority of the patients in this group were able to work without much restriction. Only two of 25 patients were prevented from working on account of their cardiac condition. It is evident that cardiac patients, with a vital capacity of 90 per cent or more of the normal, are almost always in a good state of compensation. They do not suffer from dyspnea after exertion, and if they are prevented from performing their usual task, it is usually on account of cardiac pain or other disturbances.

Group Two consists of cases in which the vital capacity is 70 to 90 per cent of the normal. A history of dyspnea on moderate exertion was a symptom usually given by these patients, but the majority could work and lead a satisfactory, though somewhat restricted life.

It may be said, in general, that cardiac patients with a vital capacity of from 70 to 90 per cent of the normal may have marked heart lesions, but usually are able to lead satisfactory, but restricted, lives. Almost all of these patients give a history of dyspnea, and have a distinctly limited cardiac reserve; they may be regarded as border-line cases in which the activities must be some-

what limited, but in which, under favorable circumstances, there is little evidence of decompensation.

Group Three consists of patients whose vital capacity is 40 to 70% of the normal. The characteristic feature of this group is the much less favorable clinical condition than those with a higher vital capacity. Dyspnea on moderate exertion was always complained of, and usually was the most prominent symptom. All patients whose vital capacity was 40 to 45 per cent of the normal were in bed. Some of them were slightly dyspneic when completely at rest, and others upon the least exertion. With the vital capacity of from 40 to 60 per cent of the normal, patients were rarely dyspneic while in bed, and most of them could walk slowly without becoming short of breath. When the vital capacity was between 60 and 70 per cent, the patients usually could walk fairly comfortably, and could even ascend the stairs without distress. Of the 67 patients comprising this group, 7 per cent could do light work, 33 per cent were up and about, and 34 per cent were in bed at the time of the examination.

Group four consists of patients whose vital capacity is 40 per cent of the normal or less. The patients of this group were compelled to remain in bed, and practically all showed signs of decompensation. Many were dyspneic when absolutely quiet, and others on the slightest exertion. In 8 patients whose vital capacity was below 30 per cent, extreme dyspnea and orthopnea were noted. The lowest vital capacity found was 17 per cent. There was a close relation between the clinical condition and the vital capacity, and as these patients improved, there was a corresponding rise in the vital capacity. Patients whose maximum respiratory exchange falls within this class during their first period of decompensation may improve so that they are able to return to a fairly normal life, but the occurrence of such a low vital capacity in the later attacks makes the prognosis unfavorable. Few patients who have at any time fallen into this group have shown great clinical improvement, and the mortality is more than 50 per cent.

It is evident, then, that there is a close relationship between the clinical condition of cardiac patients and the vital capacity of the lungs. If the maximum respiratory exchange be known, one can tell with considerable accuracy what the functional condition of the patient probably is. Decompensated patients show a low vital capacity which rises with improvement, and the extent of the increase corresponds to the degree of clinical improvement. When the vital capacity remains constant, the patient's condition remains unchanged. A rapidly rising vital capacity after a period of decompensation indicates a favorable prognosis, while a failure to rise more than a small amount or the maintenance of a continuously low vital capacity is indicative of a less favorable outlook. Slight changes in the vital capacity of ambulatory patients may be of a great significance. Peabody and Wentworth illustrate this by the case of a stained glass worker "who has a double mitral disease and auricular fibrillation. When in his best physical condition, his vital capacity is 2,600 c. c., or 65 per cent of the normal. At such times, he can walk slowly without discomfort, and can do a little light work. May 1, 1916, he came to the outdoor department, stating that he felt poorly and found that he was getting out of breath more easily than usual. His vital capacity was found to have decreased to 2000 c. c., or 50 per cent. He was given digitalis and told to go to bed for a week. At the end of this time on May 19, 1916, he reported again, to say that he was as well as before his upset, and his vital capacity had risen to 2600 c. c."

Determination of the vital capacity has been of service in correcting false impressions derived from the histories of certain patients. Neurotic women may complain of shortness of breath, which is apparently out of proportion to the physical findings in the examinations of the heart, and the vital capacity may be so high as to afford no explanation for such tendency to dyspnea. When the suspicion exists that the sympton is due to nervousness, the patient may be tested by walking rapidly and by climbing stairs. No abnormal dyspnea will result and the determination of the vital capacity will serve to

confirm the physical examination. A few patients underestimate their respiratory discomfort on exertion. The vital capacity is lower than one would expect from the history. Exercise tests will demonstrate the patient's reserve is much less than he has stated, and here again the vital capacity determination is a helpful check on the history. In other cases, in which the history of dyspnea seems out of proportion to the results of physical examination, the vital capacity may be low. In these patients, the course of the disease will confirm the value of the history and vital capacity, and shows that the physical examination gives an inadequate conception of the patient's reserve.

It is important to appreciate that changes in the vital capacity of the lungs are an index of the clinical condition only in-so-far as the cardiac weakness shows itself chiefly by producing dyspnea. This is frequently but not invariably the case. Certain patients with cardiac disease are restricted in their activities by the occurrence of palpitation or by pain rather than by becoming short of breath. The vital capacity of the lungs has no direct connection with palpitation or pain, and in cases in which these are the presenting symptoms, it does not bear any relation to the condition of the patient.

The cause of the decrease in the vital capacity of the lungs in heart disease has never been adequately explained. In advanced cases, it is due in part to pulmonary edema, pleural effusion, hepatic enlargement, and similar other factors, but in many cases, the vital capacity is decreased without any physical signs which can account for it, or with physical signs which are insufficient to explain the extent of the decrease. Siebeck suggests that this decrease in the vital capacity may be due to an engorgement or overfilling of the pulmonary vessels, and a consequent diminution of the elasticity of the lungs. Drinker, Peabody and Blumgart produced pulmonary congestion and a subsequent low vital capacity in cats by compressing the pulmonary veins at their entrance into the left auricle. From these experiments, it appears that the vital capacity may be reduced by encroachment of the dilated capillaries on the alveolar

spaces, which air could occupy under normal conditions, or the lung may be rendered less elastic through increased vascularity.

In conclusion, it may be said that we have in the vital capacity determination a test which is simple and easily performed, and at the same time, gives important information concerning the functional condition of the heart and the lungs. As an aid in diagnosis and prognosis, and to indicate the efficacy of treatment, it merits, I believe, the serious consideration of the medical world of today.

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