

CARDIAC FUNCTIONAL TESTS *

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The Eldorado of a cardiologist would be some test or combination of tests which would definitely determine the functional capacity of a given heart. This has been long sought, but is still an accomplishment for the future. The methods which have been devised and reported may be roughly classed as exercise tests, respiratory tests, tests based on the estimation of blood flow by various methods and a few other procedures.

EXERCISE TESTS IN GENERAL

It seems worth while to describe briefly the character and field of some of these, and then, in more detail, to give the results of our experience with the two which, we considered, gave promise of the best results. Taking the exercise tests first, these are based on the effects of work varying in amount all the way from that involved in rising from the supine to the upright posture to that involved in hopping or in climbing a definite number of stairs at a given rate or even in raising a 40-pound (18 kg.) weight from the floor to arm's length overhead thirty times in sixty seconds. The effects of such exercise may be measured by changes in the pulse rate, systolic blood pressure, or diastolic blood pressure, both the extent of the departure from normal and the time interval during which this is sustained being taken into consideration. The tests based on changes in systolic blood pressure seem to be more reliable than those based on changes in pulse rate. Both methods have their advocates. Both give results which vary in different persons with the type of nervous makeup and general physical condition as well as with the cardiac power or reserve. The best French authors place no reliance on either test as indicating the condition of the myocardium or of the general cardiovascular apparatus.¹ There seems to be much the same feeling in the United States, although our authors have not been so definite in their denunciation.

Combinations of pulse and blood pressure changes after exercise have been used in making ratings of physical efficiency by Schneider² and, in a different manner, by Crampton.³ Barach⁴ has arranged a so-called "energy index of the circulatory system" which takes into consideration both systolic and diastolic blood pressure and pulse rate at rest. All these tests are open to the same criticism as that which holds for the individual tests of which they are composed, and none have enjoyed much favor.

RESPIRATORY TESTS IN GENERAL

Of the respiratory tests, the most popular at present is the determination of the vital capacity, which will be discussed later. The length of time during which the breath can be held corresponds roughly to this,

and has been used as a measure of cardiac dyspnea and efficiency; but it is even more easily influenced by neurotic factors than is the vital capacity, and the differences between normal and abnormal are smaller, making the error proportionately greater. Expiratory power as well as pulmonary capacity have been measured by the height to which a column of mercury can be blown and the time during which this height can be maintained. These tests, too, have proved of but limited value.

The rebreathing apparatus, with the possibility of its clinical application as a test of cardiac efficiency, was a war product. In testing the response of aviators to high altitudes, or, what is the same thing, lowered oxygen tensions, it was found that the effects of loss of sleep, alcohol and heavy eating were readily detected by a failure of proper compensation to low tensions.⁵ This was taken to indicate temporarily lowered physical efficiency, and it was hoped that by this means the true cardiac capacity might be determined. Careful observers, however, have found that even patients with definite clinical cardiac failure do not all show poor reactions to lowered oxygen tensions.⁶ From the cardiologist's point of view, the results obtained do not justify the apparatus and time necessary to make the determination.

OTHER TESTS

Blood flow has been determined by the nitrous oxid absorption method, and has been shown to be considerably reduced in disease, as much as one half in well marked heart failure.⁷ Venous pressure determinations have given much the same information; the easiest method is that in which the minimal amount of pressure is determined which is required to collapse the veins on the back of the hand, a water manometer being used.⁸ Determinations of the oxygen and carbon dioxide content of venous and arterial blood have also yielded information regarding blood flow,⁹ but all these examinations are open to the same objections: they tell little except in cases of definite heart failure, and they require considerable technical training and apparatus.

In examining cases of "irritable heart" during the war, use was made of a large number of drugs, but none proved of much diagnostic value. Perhaps the best one was amyl nitrite, simply because its effects closely resembled those of exercise. In general, the unstable heart responded by a greater rise in rate and greater drop in blood pressure than did the normal.¹⁰ The effects on the pulse rate of a sudden flame or pistol shot were similar, and were considered by the investigators as giving some indication of functional value.¹¹

In the examination of cardiac dilatation by means of the fluoroscope after coughing, it was found that normal hearts dilate appreciably under this increase in intrapulmonary pressure, returning to normal more

5. Henderson, Yandell, and Seibert, E. G.: Organization and Objects of Research Board, Air Service, J. A. M. A. **71**: 1382 (Oct. 26) 1918. Whitney, J. L.: III. Cardiovascular Observations: Ibid. **71**: 1389 (Oct. 26) 1918.

6. Stengel, A.; Wolferth, C. C., and Jonas, L.: Breathing of Air of Lowered Oxygen Tension as a Test of Circulatory Function, Am. J. M. Sc. **161**: 781 (June) 1921.

7. Lundsgaard, Christen: Recherches sur le debit par minute du cœur, Arch. d. mal. du Cœur, Jan., 1917, p. 45.

8. Brown, N. W.: Simple Method for Determination of Venous Pressures, Bull. Johns Hopkins Hosp. **29**: 93 (April) 1918.

9. Harrop, G. A., Jr.: Oxygen and Carbon Dioxide Content of Arterial and Venous Blood in Normal Individuals and in Patients with Anaemia and Heart Disease, J. Exper. Med. **30**: 241 (Sept.) 1919.

10. Cotton, T. F.; Slade, J. G., and Lewis, Thomas: Observations on Amyl Nitrite, Heart **6**: 311, 1917.

11. Meakins, J. C., and Wilson, R. M.: Effect of Certain Sensory Excitations on the Cardiac Rate and Respiratory Rates in Cases of Irritable Heart, Heart **7**: 17, 1918.

* Read before the Boston Association of Cardiac Clinics, Boston City Hospital, March 16, 1922.

* From the Medical Clinics, Massachusetts General Hospital, and the Public Health Service Hospital, Parker Hill, Boston.

1. Gallavardin, L.: Moyens d'exploration de la capacité fonctionnelle du cœur, J. de méd. de Lyon, Aug. 20, 1920.

2. Schneider, E. C.: Cardiovascular Rating as Measure of Physical Fatigue and Efficiency, J. A. M. A. **74**: 1507 (May 29) 1920.

3. Crampton, C. W.: Proc. Soc. Exper. Biol. & Med. **12**: 119, 1915.

4. Barach, J. H.: Energy Index of Circulatory System, Am. J. M. Sc. **152**: 84 (July) 1916.

rapidly than those with a weakened myocardium.¹² Similar observations have been made following a series of blows over the course of the abdominal aorta or excitation of the skin of the precordial area, but none have proved of practical value.

From all these procedures, we selected what seemed to be the best respiratory test, the vital capacity, and the best exercise test, that in which considerable work is done with dumb-bells, and the effect on systolic blood pressure observed. We then tried to determine the sphere of usefulness of each, and the extent to which it might be trusted.

VITAL CAPACITY

The test for vital capacity consists in having the subject inhale as deeply as possible and then exhale fully into a spirometer. It represents the greatest single expiration of which the patient is capable. This has been shown to be reduced in cardiac failure of the congestive type, and, if failure is well marked, there is a reduction of more than 10 per cent. below the calculated normal in 100 per cent. of cases. And the greater the failure, in general, the lower the vital capacity.¹³ In our endeavor to determine its sphere of usefulness, we made a test of vital capacity virtually a routine in all medical ward patients, and the figures in the accompanying table and charts are based on the findings in a series of 144 such patients, forty-eight of whom had some cardiac complication, sixteen some pulmonary involvement, and eighty nothing in the history or physical examination which suggested a pathologic condition in the heart or lungs. The figures for normality used are those of West,¹⁴ based on sex and surface area and determined from healthy persons, not from ward bed patients.

The accompanying table shows that of the eighty patients with extrathoracic lesions 50 per cent. had vital capacities which were at least 20 per cent. below the calculated normal, supposedly a definitely pathologic reading, and yet those persons had no demonstrable pathologic condition in the heart or lungs. These low readings may be in part explained by the apparent effect of increasing age acting to lower the vital capacity, and by the fact that the standards seem disproportionately high for the female sex. Taking the group to which the standards of normality are best suited, namely, men below 50 years of age, there still remain, even in this group, 37 per cent. whose vital capacity is distinctly below normal limits, and yet these men had no intrathoracic lesions so far as we could tell. The diagnoses were varied, such as gastric ulcer, duodenal ulcer, pyelitis and arthritis, while other ward patients having the same diagnoses had vital capacities well

within the normal limits, so that the type of disease seems to have nothing to do with these low readings. Variable cooperation we do not believe can explain the large percentage of low readings, as many were repeated after several days and found to check, and the number of uncooperative ward patients does not approach 50 per cent. in other studies.

Of those patients with pulmonary complications for whom vital capacities were obtained, a group of sixteen, all had readings which fell below 65 per cent. of normal, the diagnoses ranging from bronchitis to convalescent lobar pneumonia. There seems to be no question that almost any pulmonary complication will lower the vital capacity, at times more markedly than moderately advanced cardiac failure.

In the cardiac group, most of those who had no congestive heart failure had normal vital capacities, while 100 per cent. of the patients with congestive failure had low vital capacities. Patients with anginal failure did not necessarily have lowered readings. The vital capacity often varied in close correspondence with the clinical improvement. For example, Chart 1 shows the change in vital capacity of a man, aged 56, who came into the hospital waterlogged, markedly dyspneic,

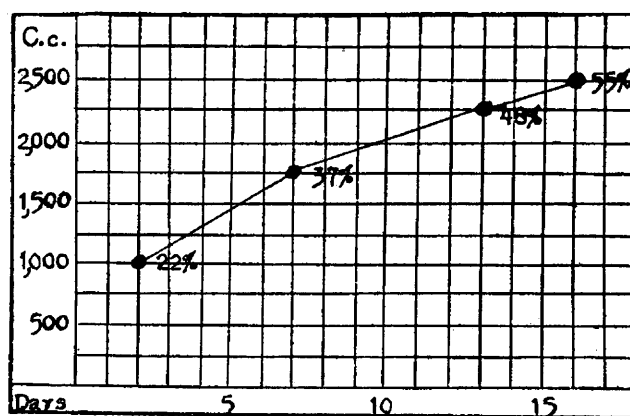


Chart 1.—Vital capacity in case of congestive heart failure, showing steady improvement.

and with a rapid ventricular rate in auricular fibrillation. He improved steadily and rapidly, just as the chart indicates. From the slowing of the heart rate, the diuresis and the loss of weight, and from the disappearance of the edema and dyspnea, we knew that he was improving and that the prognosis was good, so that following the vital capacity gave us little help in diagnosis or prognosis. It had remained stationary in some cases of less rapid clinical improvement, and it seemed often

to lag behind clinical improvement. Thus, our experience with measurement of vital capacity in cardiac patients simply confirms the work of Peabody and others, except that we would place less reliance on it in giving a prognosis or in managing a case. The case cited demonstrates what seems to be the chief

RESULTS OF VITAL CAPACITY DETERMINATIONS ON EIGHTY BED PATIENTS WITH NORMAL HEART AND LUNGS

Age	Vital Capacity	Men		Women	
		Number	Per Cent.	Number	Per Cent.
Under 50	Normal.....	24	..	7	..
	20 per cent. + subnormal	14	37	13	60
Over 50	Normal.....	7	..	2	..
	20 per cent. + subnormal	9	56	4	66
Total normal.....				Number	Per Cent.
				40	50
20 per cent. + subnormal.....				40	50

value of the test for vital capacity, namely, it furnishes a quantitative measure of dyspnea and describes it more clearly than such terms as "slight," "moderate" or "marked," since the personal equation enters into these to a great extent. When cardiac failure is proportional to dyspnea, vital capacity would be propor-

12. Guthrie, J. B.: Cough-Dilatation Time a Measure of Heart Function, J. A. M. A. **62**: 30 (Jan. 3) 1914.

13. Peabody, F. W.: Vital Capacity of the Lungs in Heart Disease, Med. Clin. N. Am. **4**: 1655 (May) 1921.

14. West, H. F.: A Comparison of Various Standards for the Normal Vital Capacity of the Lungs, Arch. Int. Med. **25**: 306 (March) 1920.

tional to the failure, provided the standards were suitable to bed patients. We do not believe this to be the case, at least so far as hospital ward patients are concerned. Vital capacity, to our minds, adds to the clarity of the records, but rarely aids in the actual management or "sizing up" of a case.

EXERCISE TEST

The exercise tests are even less satisfactory. Selection was a more difficult problem; but the test which seemed to give most promise was this: The patient, if an adult, swings two 20-pound (9 kg.) dumb-bells from the floor to arm's length overhead ten, fifteen and twenty times, allowing from two to three seconds for each complete swing. Systolic blood pressure is then taken as soon as possible, usually within twenty-five seconds, and it is followed at from ten to fifteen second intervals until it is definitely dropping, then at thirty second intervals until it reaches within 6 mm. of the preexercise level of mercury. There is normally a sharp rise in systolic blood pressure within the first forty seconds after work, followed by a fall to within 6 mm. of normal within two minutes. If a healthy person continues to work at the rate described until he has nearly reached his limit of capacity, the maximum systolic blood pressure will not be obtained until from sixty to eighty seconds after stopping work instead of the normal forty seconds, and the pre-exercise level will not be reached until the end of from three to six minutes instead of two. Such a curve is said to have a "delayed rise" or "delayed summit" and "prolonged fall."¹⁵

Chart 2 illustrates the two types of curve that are obtained. The lower one, which is a normal curve, was produced by a cardiac patient without heart failure. The upper curve, which presents a "delayed rise," was given by a patient with effort syndrome.

The two men were of about the same age and size, and they did the same amount of work, swinging two 20-pound dumb-bells, twenty times in sixty seconds from floor to overhead. Such a "delayed rise" occurs only when the person has nearly reached his limit, and is accompanied by fatigue, hyperpnea and, at times, palpitation, dyspnea, flushed face and the like. It never occurs unless the patient is near the end of his strength, and for this reason has been taken to represent an overtaxing of the cardiac capacity or reserve. Its sphere as applied to patients is obviously limited. It should not be used in cases with definite heart failure or in those showing evidence of infection. In cases without heart failure, however, it may serve, with good reason, as a basis for advice as to the amount of exercise which may be taken with safety. There would seem to be little need of restricting the exercise of a

patient who can swing 40 pounds (18 kg.) from floor to overhead, twenty times in forty seconds, without a delayed rise in blood pressure, no matter what may be heard when listening to the heart.

Our experience has been limited to twenty-nine cases, these being divided into three groups, ten normal persons, ten with more or less marked effort syndrome, and nine with organic heart disease, all free from heart failure and evidence of recent infection. We made the mistake at first, which others¹⁶ have made, of starting with weights which were too light, using two 5-pound (2.2 kg.) dumb-bells. With these, the arm muscles of the patients became tired from frequent motion long before they had done enough actual work to cause any marked changes in blood pressure. This is similar to the situation found occasionally in children when simple muscular weakness forces the child to stop before a delayed rise in blood pressure can be obtained.¹⁷ It was overcome in adults by the use of larger weights, most of the tests consisting in swinging two 20-pound (9 kg.) dumb-bells from floor to overhead, fifteen and, later, twenty times in forty and sixty seconds, respectively.

Using this routine, it can be said that the work with

our normal patients confirms that of the advocates of this test. Delayed rises were obtained on nearing the maximum exertion and not before. Work with the effort syndrome cases, however, was less satisfactory. Two patients showed blood pressures which changed so readily from second to second, with variations as great as 10 mm. of mercury, that no interpretation could be placed on the curves. Others showed a dissociation between the two features "delayed summit" and "prolonged fall," having none of the former,

but definitely "prolonged falls" of three and a half and four minutes, or vice versa. Such curves also are difficult to interpret.

The cardiac patients with organic lesions showed essentially the same features. In both classes of patients, the "delayed rise" seemed to depend very much more on general muscular condition than on what might be guessed as their true "cardiac reserve"; two patients with effort syndrome with apparently normal cardiac musculature showed a delayed rise on fifteen swings of 40 pounds (18 kg.), while two patients with organic cardiac lesions did not show it until after performing twenty swings. Others have found this to be true, also, but have laid little stress on it. It prevents any reliance being placed on the test for diagnostic or prognostic purposes generally, so far as the heart is concerned, and confines its usefulness simply to following the individual case, even there realizing that the patient's general condition is being followed and not the cardiac condition. It is perfectly possible to imagine a man's exercise tolerance rising

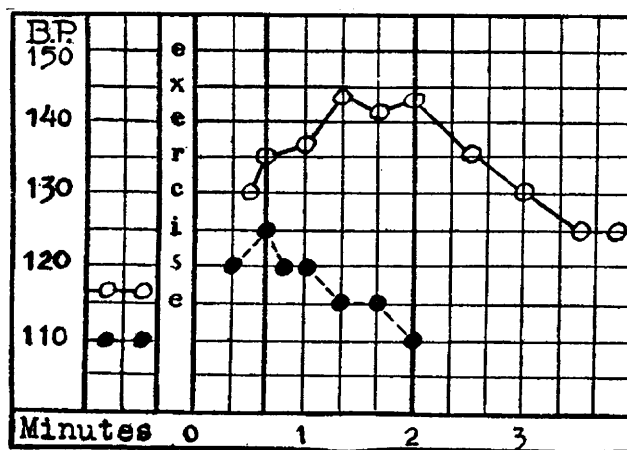


Chart 2.—Blood pressure response to exercise: normal curve indicated by heavy dots and broken line; delayed rise indicated by rings and solid line.

15. Barringer, T. B., Jr.: Studies of the Heart's Functional Capacity, *Arch. Int. Med.* **20**: 829 (Dec.) 1917. Cotton, T. F.; Rapport, D. L., and Lewis, Thomas: After-Effects of Exercise on Pulse Rate and Systolic Blood Pressures in Cases of Irritable Heart, *Heart* **6**: 269, 1917.

16. Mabon, T. McC.: Studies of Cases of "Effort Syndrome" with Measured Work, *Am. J. M. Sc.* **158**: 818 (Dec.) 1919.

17. Wilson, May G.: Exercise Tolerance of Children with Heart Disease, *J. A. M. A.* **76**: 1629 (June 11) 1921.

through improvement in general muscular condition as a result of daily exercise, while his cardiac condition remains unchanged or even becomes worse.

This lack of specific information given by the test, the difficulties in interpreting many curves, together with the fact that there are no good standards for normality for adults, and the small number of cases to which the test is applicable make it, in its present form, an unsatisfactory test of cardiac efficiency.

The results of the vital capacity and exercise tolerance tests of our normal persons are in good agreement; but this does not hold true with our pathologic cases, if the previously accepted standards for vital capacity are used. Two cardiac patients had vital capacities of only 62 and 68 per cent. of the calculated normal, and yet very nearly normal exercise tolerance tests. Both were able to swing 40 pounds (18 kg.), fifteen times in forty seconds without showing a "delayed rise" or "prolonged fall" in blood pressure, and these were of small degree after twenty swings with 40 pounds (18 kg.) in sixty seconds. On the other hand, one cardiac patient with a vital capacity of 93 per cent. of the normal showed a marked "delayed rise" in blood pressure after swinging 40 pounds (18 kg.) fifteen times, as was also shown by a patient with effort syndrome with a vital capacity of 110 per cent. of the normal.

SUMMARY

1. There are numerous cardiac functional tests. Most of them are of small value. Two have been selected for further study.

2. Vital capacity determinations add to the exactness of records but give little aid in the clinical study of a patient. We have found that 50 per cent. of ward patients with no demonstrable pathologic condition in the heart or lungs have vital capacities of 20 per cent. and more below the so-called normal.

3. The exercise tolerance test described is applicable to a limited type of case, and even there gives little information of the actual cardiac condition.

4. Vital capacity and exercise tolerance determinations were markedly at variance in 14 per cent. of the cases in which both tests were performed.

5. There is no satisfactory test of cardiac functional capacity at the present time.¹⁸

18. Judgment in the interpretation of the general reaction of an individual to the habitual activities of his or her life (such as the ascent of stairs) affords the physician more valuable information, as a rule, about the circulation than the use of any of the functional tests described.

Vital Statistics.—One of the most important subjects that can come before a state legislature is the passage of wise and efficient laws for the collection, preservation and compilation of proper and accurate vital statistics. . . . In the last analysis, human life and its perpetuation is the predominant factor in all problems, whether personal, social, state or national. The standing of a nation, ultimately, is only to be measured by the standard of human lives. These general principles have long been admitted by all statesmen and economists. It comes as somewhat of a shock to one to realize that in a nation where a record is made of every legal procedure, of every business transaction and commercial liability, no matter how insignificant, where millions are spent each year in recording and preserving all real estate transactions, where everything else, material or immaterial, is made a matter of record; yet in more than half of the United States a human being can be born and die without any record being made or official notice being taken of the fact. Elaborate systems record the birth, career and death of every pedigreed horse, cow, dog, and even of "blood cats," yet children are born and men and women die without record of these events being preserved.—*A. M. A. Bull.* 4:106, 1909.

THE MINOR FORMS OF PULMONARY EMBOLISM AFTER ABDOMINAL OPERATIONS

A CLINICAL AND ROENTGENOLOGIC STUDY *

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During the last few years the opinion that embolism is a more important factor in the production of post-operative pulmonary complications than has heretofore been suspected has gradually been gaining ground. Since 1920, representatives of several of the largest surgical clinics in America and Europe have accepted this conclusion (Boland,¹ Farrar,² Cutler and Hunt³). In January, 1922, DeQuervain⁴ of Switzerland stated that following operations upon the stomach, "three fourths of the true postoperative deaths are due to lung complications—emboli, pneumonia and lung gangrene," and that in a good share of the cases of so-called pneumonia, the processes are really embolic in nature.

We⁵ arrived at this conclusion in 1919, after having studied all the postoperative pulmonary complications and cases of femoral thrombophlebitis that had occurred in the gynecologic service of the Johns Hopkins Hospital during the preceding thirty years. At that time we expressed the opinion that 40 per cent. of our cases that had been diagnosed as pleurisy, and 12 per cent. of those diagnosed as bronchopneumonia or pneumonia had been in reality instances of pulmonary infarction and hence were due to pulmonary embolism. Moreover, we pointed out the clinical features on the basis of which we attempted to distinguish the embolic from the inflammatory pulmonary complications and laid stress on the importance of making this differentiation. This report was published in 1920.

There still seems to be a great deal of confusion concerning this subject. Certain observers have gone so far as to state that complications which have been considered inflammatory and irritative phenomena are due to showers of miliary emboli and that this is the cause of many of the cases of postoperative pneumonia. Others are apparently content with a noncommittal attitude, admitting that some of the cases heretofore diagnosed as pneumonia may be due to embolic processes, but making no effort to distinguish them in actual practice. In our experience, embolic phenomena present a clinical picture which is usually characteristic and altogether different from that found in pneumonia and bronchopneumonia; we also believe that the potential and actual importance of even minor embolic complications of the lung demand that we attempt to recognize them and institute the proper treatment.

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* Read before the Section on Obstetrics, Gynecology and Abdominal Surgery at the Seventy-Third Annual Session of the American Medical Association, St. Louis, May, 1922.

1. Bolland, F. K.: Postoperative Thrombophlebitis, *South. M. J.* 45: 307-310 (April) 1921.

2. Farrar, L. K. P.: The Incidence of Pulmonary Embolism and Thrombosis Following Hysterectomy for Myoma Uteri, *Am. J. Obst. & Gynec.* 2: 286-296 (Sept.) 1921.

3. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, *Arch. Surg.* 1: 114-157 (July) 1920.

4. DeQuervain, F.: A Consideration of the Relative Merits of Resection and Gastro-Enterostomy in the Treatment of Gastric and Duodenal Ulcer, *Surg., Gynec. & Obst.* 34: 3 (Jan.) 1922.

5. Hampton, H. H., and Wharton, L. R.: Venous Thrombosis, Pulmonary Infarction and Embolism Following Gynecological Operations, *Bull. Johns Hopkins Hosp.* 31: 95-117 (April) 1920.