We have followed the foregoing routine in over 200 cases, and I feel justified from the uniformity of our observations in stating that if the test shows the immune mechanism to be intact, operative treatment is indicated. Conversely, if the test shows the immune mechanism to be destroyed, operation is useless except for palliative reasons.

Again, I believe that operation is best performed when the percentage of large mononuclear cells is high, as metastasis is less liable to occur from the operative handling if the anticancer ferment is active when the operation is performed.

One more word concerning the value of this test: It is a valuable prognostic aid. After complete destruction of the immune mechanism, which may occur occasionally when the initial tumor is small, the progress of the disease is always rapid, and operation in such cases is usually followed by rapid general metastasis, while many cases, apparently far advanced, will respond to stimulation of the immune mechanism. Such patients always live much longer than the physician anticipates, unless he is particularly guarded in his prognosis. It is this class of case, with the alternating periods of retrogression and advancement of growth, that furnishes the fruitful field for the work of the charlatan, since the temporary benefit observed from Nature's efforts is usually attributed to whatever medication is being used at the time.

ABSTRACT OF DISCUSSION

DR. HENRY SCHMITZ, Chicago: I made the assertion some time ago, and again here in discussing Dr. Levin's paper, that in the study and treatment of carcinoma we should take into consideration the leukocytic and the differential white blood corpuscle count. By these the value or uselessness of different therapeutic measures could be shown. If, for example, following the use of a certain therapeutic measure, there were a decrease in the red and the white blood count, we should wait until the blood returned to normal before making another attempt to treat the patient. If the decrease persists, further treatment would be useless. This is specially true in the radium and roentgen treatment of cancer. Persistence of leukopenia indicates the hopelessness of the treatment. Additional exposures to the rays merely render the patient and the tumor worse. I therefore feel that the observations of Dr. Vaughan are of inestimable value, not only in the determination of operability, and the prognosis of surgical treatment of carcinoma, but also of the palliative treatment. On my return home I shall immediately begin a study of my work from Vaughan's standpoint and see whether I can substantiate his deductions.

DR. SAMUEL W. BANDLER, New York: I am delighted to hear the word immunity introduced in the study of carcinoma. I believe the preventive study will be along the line of immunity—determining why some of us have it and others do not. It would be interesting also in families to study whether the tendency to carcinoma is a characteristic of more than one member of the family or only of the person who has the disease. I have known histories in which the father has had carcinoma, in which three children have died in infancy; one adult child of that father has tuberculosis; another adult daughter has carcinoma of the stomach at the age of 37; another adult daughter has carcinoma of the colon at the age of 34. Circumstances such as that point absolutely to a lack of resistance in that family, which takes the form of carcinoma in the majority, of tuberculosis in one, and death in infancy or childhood in the others. It would be interesting to see in these cases whether there is a familial change in the blood and in the relation of the cells in other members of the family than the one with carcinoma. I would ask whether Dr. Vaughan has made studies of the same sort in benign tumors of various parts of the body, pointing to immunity

in one individual and a lack of it in another. In women who have been pregnant we find in a certain proportion the development of hydatid mole, or an overgrowth of cells which are supposed to atrophy and disappear, showing a lack of protection or resistance in that individual.

DR. J. WALTER VAUGHAN, Detroit: The only studies we made with benign tumors were in making extracts from the tumors and testing out their power to increase the percentage of large mononuclear cells. The more the tissue cells revert to embryologic type, the higher the increase—from 6 to 10 per cent. I am glad that the point has been brought out that the repeated differential blood count is of the utmost importance and gives us more aid clinically than any other examination.

RELATION OF VITAL CAPACITY OF LUNGS TO CLINICAL CONDITION OF PATIENTS WITH HEART DISEASE *

CHARLES W. McCLURE, M.D.

AND

FRANCIS W. PEABODY, M.D.

BOSTON

In most persons with heart disease, dyspnea is one of the earliest symptoms observed, and the tendency of a patient to become short of breath on exertion bears a close relation to his clinical condition. The physician often asks of his patient, "Do you get short of breath easily?" or "Do you get short of breath more easily than you did formerly?" and thereby indicates that he regards the question of dyspnea as having a direct bearing on the functional state of the heart. An increasing tendency to dyspnea is evidence of a failing cardiac reserve, while the ability to perform a greater amount of exercise without becoming short of breath indicates, in general, an improvement in the efficiency of the heart. In a limited number of patients, palpitation or cardiac pain are the presenting symptoms, and dyspnea does not assume so prominent a rôle, but in a much larger group the onset of respiratory distress is the first symptom which suggests that the subject is approaching the limit of the functional capacity of his heart.

On account of the importance of the tendency to dyspnea as an index of the clinical condition of the patient, we not infrequently attempt to standardize it or describe it in our records, so that we may know more or less accurately whether it is increasing, decreasing or remaining constant. Unless, however, careful observations are made on the patient while he is undergoing graduated exercises, one has little but the patient's own statement to rely on, and such observations concerning subjective symptoms are, of course, apt to be extremely vague and unreliable. It would be of considerable assistance, therefore, if we could devise some objective method of measuring accurately the tendency to dyspnea. The latter it seems possible to do in the determination of the vital capacity of the lungs.

As a result of a study of the various elements which take part in the production of dyspnea in heart disease, it was found that perhaps the most significant feature was the inability to breathe deeply. When a person

* From the Medical Service of the Peter Bent Brigham Hospital, and the Department of Medicine, Medical School of Harvard University.

* Read before the Section on Practice of Medicine at the Sixty-Fifth Annual Session of the American Medical Association, New York, June, 1917.

exerts himself, as by walking or running, the metabolism becomes increased, the oxygen consumption is raised, and in order to meet the need for additional oxygen, the rate and depth of respiration are increased. The normal subject accomplishes this without difficulty, since he can easily breathe many times deeper than he does while he is at rest; but for the patient with severe heart disease, it is quite a different problem, for the amount to which he can increase the depth of his respiration is much restricted. If one measures the vital capacity of the lungs of such a patient, and this is the volume of air which he can expire after the deepest possible inspiration, it will be found to be much below the normal. The importance of this inability to breathe deeply, which manifests itself as a decrease in the vital capacity of the lungs, is shown still further by the fact that there is a close relationship between the degree of the decrease in vital capacity and the degree of the tendency to dyspnea. Observations reported elsewhere on 124 patients¹ and

1. Peabody and Wentworth: Arch. Int. Med., September, 1917, p. 443.

TABLE 1.—VITAL CAPACITY DURING PROGRESS OF CARDIAC DISEASE

Case	Date	Vital Capacity		Condition	Remarks
		C.c.	%		
1 Med. No. 6165 Chronic myocarditis Auricular fibrillation	2/20	1,825	65		Comfortable while at rest in bed. Lungs clear
	2/21	1,875	66	Stationary	Subjective improvement
	2/24	1,825	65	Stationary	
	2/27	1,800	68	Stationary	
	3/1	1,500	53	Worse	Hydrothorax has developed since last observation
	3/4	1,600	56	Stationary	Hydrothorax present
	3/5	1,500	56	Stationary	Hydrothorax present
	3/12	1,525	56	Stationary	Hydrothorax present
	3/16	1,500	56	Stationary	Hydrothorax present
	4/4	1,450	51	Stationary	Hydrothorax present
	4/6	1,300	50	Stationary	
	4/10	1,250	44	Worse	Increased dyspnea
	4/19	1,000	35	Worse	Dyspnea and cough
	4/20	1,200	42	Stationary	
	4/23	1,400	50	Improved	Dyspnea has gone; no pulse deficit
	4/30	1,200	42	Stationary	
	5/11	1,400	50	Stationary	
	5/17	1,400	50	Stationary	No change since April 23; still in bed
2 Med. No. 6274 Chronic myocarditis	3/13	1,700	35		Badly decompensated anasarca
	3/14	1,900	39	Stationary	
	3/16	1,900	39	Stationary	
	3/19	1,700	35	Stationary	
	3/22	1,900	39	Stationary	
	3/26	2,000	42	Improved	Less edema of legs; less dyspnea
	3/31	1,950	41	Stationary	
	4/4	2,050	43	Stationary	
	4/6	2,000	42	Stationary	
	4/10	2,300	48	Improved	Less edema of legs; less hydrothorax
	4/17	2,275	47	Improved	Less edema of legs
	4/20	2,500	46	Stationary	
	4/23	2,400	50	Stationary	
	4/27	2,500	52	Stationary	
	4/30	2,400	50	Stationary	
	5/2	2,500	52	Stationary	Slow but steady improvement occurred throughout stay in hospital
	5/5	2,400	50	Stationary	
	5/7	2,400	50	Stationary	
	5/9	2,500	52	Stationary	
	5/15	2,500	52	Stationary	
	5/17	2,700	56	Improved	Slight edema of shins; right hydrothorax; out of bed but very much restricted
3 Med. No. 6194 Chronic myocarditis	3/5	1,850	46		Badly decompensated anasarca
	3/7	1,800	48	Stationary	Badly decompensated anasarca
	3/14	2,000	50	Stationary	Badly decompensated anasarca
	3/26	1,600	40	Worse	Badly decompensated anasarca; more dyspnea
	4/4	1,800	45	Stationary	Anasarca
	4/10	1,575	39	Worse	Anasarca; more dyspnea
	5/5	1,600	40	Stationary	Anasarca
	5/10	1,800	45	Stationary	Anasarca; still in bed

TABLE 1.—VITAL CAPACITY DURING PROGRESS OF CARDIAC DISEASE—Continued

Case	Date	Vital Capacity		Condition	Remarks
		C.c.	%		
4 Mitral stenosis Tricuspid insufficiency	2/10	2,100	43		Enlarged liver; acutely decompensated; anasarca
	2/12	2,130	45	Stationary	
	2/14	3,000	62	Improved	Less ascites; liver less tender
	2/19	3,500	73	Improved	Liver not tender
	2/27	3,100	65	Worse	Slight increased dyspnea; dry cough
	3/1	2,575	54	Worse	Increased dyspnea; liver slightly tender; increased ascites
	3/3	2,700	56	Stationary	
	3/5	2,400	50	Stationary	
	3/8	2,200	46	Stationary	No change clinically
	3/12	2,650	55	Improved	Cough stopped and edema is less
	3/16	3,100	64	Stationary	
	3/19	2,750	53	Stationary	
	3/22	2,700	56	Stationary	
	3/26	2,500	52	Stationary	
	3/31	2,800	58	Improved	No dyspnea; less ascites
5 Med. No. 6317 Chronic myocarditis	3/23	2,000	41		Ascites; hydrothorax; orthopnea
	3/24	1,950	40	Stationary	
	3/26	2,200	46	Improved	No ascites; less dyspnea
	3/31	2,200	46	Improved	Less edema; no hydrothorax
	4/4	2,150	45	Stationary	
	4/6	2,200	46	Stationary	
	4/10	2,300	48	Stationary	
	4/17	2,500	52	Stationary	
	4/23	2,800	58	Improved	Signs unchanged, but looks better
	4/27	2,700	56	Stationary	
	5/2	2,500	52	Stationary	
	5/5	2,100	44	Worse	Edema of legs; liver large and tender
	5/7	2,300	48	Stationary	
	5/9	2,300	48	Stationary	Left hospital, but gets short of breath very easily

many subsequent observations confirm this fact. If the volume of the vital capacity is expressed in terms of the percentage of normal values determined according to sex and height,² it has been found that the following generalizations are approximately true: Cardiac patients with a vital capacity of 90 per cent. or more of the normal standards have no more tendency to dyspnea than is to be found in a similar group of healthy persons. Cardiac patients with a vital capacity of from 70 to 90 per cent. of the normal almost invariably give a history of becoming short of breath on moderate exertion, but most of them can lead a satisfactory although somewhat limited life. Patients with heart disease who have a vital capacity which is only from 40 to 70 per cent. of the normal are much more restricted. Practically all become short of breath easily, and few are able to do even the lightest work. They are borderline cases and frequently become acutely decompensated. Those patients with a vital capacity which is below 40 per cent. of the normal standard are acutely decompensated, usually confined to bed, and are often dyspneic even while at complete rest.

It having been shown that there is a direct relation between a decrease in the vital capacity of the lungs and the clinical condition of cardiac patients even when one compares the vital capacity of the individual case to more or less arbitrarily chosen normal standards, it seemed worth while to find out whether this relation between vital capacity and clinical condition would not be even more striking if frequent observations were made in the same case. In a considerable

2. The normal values for the vital capacity of the lungs are: Males: Height from 159.5 to 173.5 cm., 4,000 c.c.; from 173.5 to 182.5 cm., 4,800 c.c.; 182.5 cm. and above, 5,100 c.c. Females: Height from 154.5 to 162 cm., 2,825 c.c.; from 162 to 167 cm., 3,050 c.c.; 167 and above, 3,275 c.c.

group of cases, therefore, we have attempted to plot the variations in the vital capacity of the lungs and to compare with these the changes in the clinical condition. Each observation has consisted of a careful clinical examination of the patient, together with the recording of a statement as to whether the examiner

TABLE 2.—EFFECT OF EDEMA ON VITAL CAPACITY

Case	Date	Vital Capacity		Condition	Remarks
		C.c.	%		
6 Med. No. 6229 Mitral stenosis Auricular fibrillation	3/ 3	1,900	47		Orthopnea; cyanosis; edema of legs
	3/ 4	1,800	45	Improved	Less edema of legs
	3/ 5	1,900	47	Improved	Less edema and has dyspnea
	3/ 7	2,300	56	Improved	Less dyspnea; less edema of legs
	3/ 9	2,500	62	Improved	Improved; breathing easier
	3/12	2,400	60	Stationary	
	3/16	2,200	55	Stationary	
	3/19	2,400	60	Stationary	Some edema of legs; out of bed, but becomes dyspneic on slight exertion
	3/26	2,300	58	Stationary	

TABLE 3.—EFFECT OF HYDROTHORAX AND ASCITES ON VITAL CAPACITY

Case	Date	Vital Capacity		Condition	Remarks
		C.c.	%		
7 Med. No. 6284 Mitral stenosis Aortic regurgitation	3/15	2,250	44		Orthopnea; ascites; large liver; edema of legs
	3/16	2,500	..	Stationary	Less edema of legs
	3/19	2,700	53	Improved	
	3/22	2,500	49	Stationary	
	3/26	2,500	49	Stationary	
	3/31	2,900	57	Improved	Lungs clear; ascites gone; no edema
	4/ 4	2,900	57	Stationary	
	4/ 6	2,900	57	Stationary	
	4/10	2,950	58	Stationary	
	4/14	3,100	61	Stationary	Left hospital in rather poor condition; can walk, but probably unable to do work
8 Med. No. 6114 Chronic myocarditis	2/12	1,510	31		Orthopnea; edema of legs; hydrothorax; Cheyne-Stokes respiration
	2/13	1,575	33	Improved	Less dyspnea
	2/15	1,675	35	Improved	Edema gone from legs
	2/16	1,900	40	Improved	Much more comfortable
	2/19	1,800	37	Stationary	
	2/23	1,800	37	Improved	No râles in lungs; no edema of feet
	2/27	2,000	42	Improved	Cough gone
	3/ 1	2,100	45	Stationary	
	3/ 5	1,950	41	Stationary	Nocturnal dyspnea persists; in wheel chair, but condition very poor
9 Med. No. 6335 Chronic myocarditis	3/27	1,200	25		Orthopnea; much ascites and edema
	3/31	1,700	35	Improved	Condition improved; no ascites
	4/ 4	2,300	48	Improved	Lies flat without discomfort
	4/ 6	2,000	42	Stationary	
	4/10	2,250	44	Stationary	
	4/14	2,300	48	Stationary	Left the hospital compensated; patient must lead a restricted life
10 Med. No. 6344 Chronic myocarditis	3/26	2,000	50		Orthopnea; massive edema; much ascites
	3/31	2,100	53	Improved	Record of vital capacity is unsatisfactory; less ascites
	4/ 4	2,900	73	Improved	No edema of legs; ascites gone
	4/ 6	2,800	70	Improved	Moderate diuresis, no change in clinical condition
	4/10	3,000	75	Stationary	
	4/14	3,000	75	Stationary	Left the hospital

TABLE 3.—EFFECT OF HYDROTHORAX AND ASCITES ON VITAL CAPACITY—Continued

Case	Date	Vital Capacity		Condition	Remarks
		C.c.	%		
11 (Chart 1) Med. No. 6261 Chronic myocarditis Auricular fibrillation	3/ 9	1,400	42		Acutely decompensated; hydrothorax; orthopnea; edema of legs
	3/10	1,475	45	Improved	Less dyspnea but still orthopneic
	3/12	1,800	55	Improved	Less edema; less dyspnea; less hydrothorax
	3/16	2,500	76	Improved	Lungs clear; less dyspnea; hydrothorax gone
	3/19	2,700	82	Stationary	
	3/22	2,775	85	Stationary	
	3/26	2,800	86	Stationary	Left hospital in good condition
12 (Chart 2) Med. No. 6152 Mitral stenosis Auricular fibrillation	2/16	1,150	29		Orthopnea; cyanosis; anasarca
	2/17	1,200	30	Worse	Increased edema
	2/19	1,050	26	Stationary	Marked dyspnea; condition bad
	2/23	650	14	Worse	Died
13 Med. No. 6178 Chronic myocarditis Auricular fibrillation	2/20	1,400	29		Orthopnea; marked ascites; anasarca
	2/21	2,300	48	Improved	Much less dyspnea; edema unchanged
	2/22		..		Abdominal paracentesis
	2/23	2,500	52	Improved	Less dyspnea; still in bed
14 Med. No. 6541 Chronic myocarditis	5/ 5	1,500	38		Orthopnea; moderate ascites; moderate edema of legs
	5/ 7	2,150	54	Improved	Less dyspnea; no edema and no ascites
	5/ 9	3,500	88	Improved	No dyspnea
	5/16	3,600	90	Stationary	Out of bed and in good condition

considered the patient's general condition to be improved, worse or stationary, and then the determination of the vital capacity of the lungs. Instead of the recording spirometer which was used in the previous observations on the vital capacity of the lungs, in this series of cases we have used an ordinary well-balanced spirometer of 8 liter capacity. The subjects were told to take as deep an inspiration as possible, and then to insert the tube to the spirometer into the mouth and give a complete expiration. The volume of the expiration was measured on the spirometer wheel. In a few subjects who were very ill or were for other reasons unable to cooperate, and in a few who were unwilling to cooperate, satisfactory results could not be obtained, but in most instances it proved to be comparatively easy to get records that apparently represented with considerable accuracy the vital capacity of the lungs.

Judgment as to the clinical condition of our patients has been based on the degree of dyspnea, edema, pleural transudate, ascites, pulmonary congestion, acute enlargement of the liver, cyanosis, and pulse rate or pulse deficit. In two cases, of which special mention will be made, precordial pain and cutaneous or muscular hyperesthesia were considered as symptoms of myocardial weakness.

Twenty-four patients with some severe form of cardiac disease have been studied. In five patients in whom the objective evidences of myocardial insufficiency were marked, the alterations in clinical condition took place extremely slowly. For this reason it was frequently difficult or even impossible to note any changes in observations made at short intervals. It was under these circumstances that the greatest discrepancies between the vital capacity records and our

judgment of the progress of the case have occurred. Nevertheless these discrepancies have never been great, and the subsequent course of the case has invariably shown an error in judgment rather than an error in the indications of the vital capacity records. A comparison of our clinical observations as to the progress of the case with the vital capacity findings is given in Table 1.

Diminution in edema of the extremities resulting from rest in bed does not necessarily represent improvement in the functional condition of the myocardium. It may be explained as a static phenomenon resulting from a change from the vertical position assumed in sitting or standing to the horizontal position in bed. On the other hand, edema, while apparently leaving the body, may merely be redistributed, as is sometimes shown by the failure of the patient to lose weight (Table 2).

Two frequent complications of cardiac disease which modify the vital capacity are hydrothorax and

TABLE 4.—EFFECT OF ANGINOID PAIN OR CUTANEOUS AND MUSCULAR HYPERESTHESIA ON VITAL CAPACITY—Continued

Case	Date	Vital Capacity		Condition	Remarks
		O.c.	%		
20 Med. No. 6135 Mitral stenosis	2/14	1,200	42		Much dyspnea even when at rest
	2/15	1,275	45	Improved	Less dyspnea
	2/17	1,400	49	Improved	Slight edema
	2/18	1,200	42	Worse	More dyspnea
	2/24	1,400	49	Improved	Less dyspnea; comfortable in bed
	2/27	1,475	52	Stationary	
	3/1	1,400	49	Stationary	
	3/4	1,650	58	Stationary	
	3/5	1,575	55	Stationary	Up in chair; dyspnea on slight exertion
21 Med. No. 6491 Mitral stenosis	4/25	1,900	68		Dyspnea on exertion, but comfortable in bed
	4/26	1,800	64	Stationary	
	4/27	1,950	69	Stationary	
	4/30	1,950	69	Stationary	
	5/2	2,000	71	Stationary	
	5/9	2,100	74	Stationary	
	5/15	2,200	77	Improved	Less dyspnea on exertion
	5/18	2,200	77	Stationary	Out of bed; no dyspnea while walking about ward
22 Med. No. 6241 Chronic myocarditis Auricular fibrillation	3/4	3,100	78		Dyspnea; cyanosis
	3/5	3,500	88	Improved	Much better; less dyspnea
	3/8	3,500	88	Stationary	
	3/12	3,475	87	Stationary	
	3/16	3,600	90	Stationary	
	3/19	3,500	88	Stationary	
	3/22	3,500	88	Stationary	No dyspnea when walking
23 Med. No. 6273 Chronic myocarditis	3/13	2,475	51		Slight edema of shins; dyspnea; cough
	3/14	2,400	50	Stationary	
	3/16	2,300	48	Stationary	
	3/19	2,500	52	Improved	No edema of shins; much cough
	3/22	2,300	48	Stationary	Less cough
	3/26	2,600	54	Stationary	Much cough; râles in both lungs; no noticeable change in condition since entering hospital
24 Med. No. 6399 Mitral stenosis	4/7	2,500	63		Slight dyspnea at rest; bloody sputum
	4/10	3,100	78	Improved	No dyspnea at rest; no sputum
	4/17	3,400	85	Stationary	
	4/20	3,200	80	Stationary	
	4/23	3,500	88	Stationary	
	4/27	3,400	85	Stationary	Out of bed
	5/5	3,200	80	Stationary	Condition unchanged
	5/7	2,800	70	Worse	Increased dyspnea
	5/9	3,000	75	Stationary	
	5/15	3,100	78	Stationary	
	5/17	3,400	85	Improved, stationary	
	5/23	3,400	85	Stationary	Left hospital in good condition

TABLE 4.—EFFECT OF ANGINOID PAIN OR CUTANEOUS AND MUSCULAR HYPERESTHESIA ON VITAL CAPACITY

Case	Date	Vital Capacity		Condition	Remarks
		O.c.	%		
15 Med. No. 6176 Mitral stenosis	2/21	2,450	80		Cyanosis; frequent cough; slight edema; no dyspnea
	3/1	2,950	97	Improved	No edema; less cough
	3/5	2,200	72	Worse	Precordial pain
	3/9	2,700	89	Improved	No pain; left hospital in good condition
16 Med. No. 6480 Mitral stenosis	4/20	2,000	61		Dyspnea on exertion; comfortable in bed
	4/27	1,900	60	Stationary	
	4/30	2,400	73	Improved	Subjective improvement
	5/2	2,600	80	Stationary	
	5/9	2,000	61	Worse	Cutaneous and muscular hyperesthesia
	5/10	2,100	64		Hyperesthesia unchanged
	5/14	2,965	91	Improved	Hyperesthesia gone
	5/15	3,000	91	Stationary	
17 Med. No. 6262 Mitral stenosis Aortic insufficiency	5/18	3,000	91	Stationary	
	5/25	3,100	94	Stationary	Condition good; walking about the ward
	3/9	1,100	39		Orthopnea on slight exertion; moderate edema of legs
	3/10	1,200	42	Improved	Less edema
	3/12	1,375	48	Improved	
	3/16	1,650	58	Stationary	Vital capacity is increased, but no change noted in clinical condition; still in bed
	3/19	1,500	53	Stationary	
	3/22	1,850	65	Improved	Less cyanosis; less dyspnea; no edema
18 Med. No. 6293 Chronic myocarditis	3/26	1,900	66	Stationary	Gets dyspneic on moderate exertion
	4/7	1,900	48		Acutely decompensated tender, pulsating liver; no dyspnea while in bed
	4/10	2,400	60	Improved	Liver not palpable
19 (Chart 3) Med. No. 6130 Chronic myocarditis Auricular fibrillation	2/13	1,700	42		Marked dyspnea; orthopnea; much edema of legs
	2/14	2,175	54	Improved	Less dyspnea; less edema
	2/16	2,800	70	Improved	Less edema; less dyspnea
	2/19	3,200	80	Improved	Less edema; less dyspnea
	2/23	3,150	79	Stationary	
	2/27	2,550	88	Stationary	Less cough
	3/1	3,550	88	Improved	
	3/5	3,200	80	Stationary	
	3/8	3,450	86	Stationary	
	3/12	3,575	89	Stationary	General condition good; walking about the ward

ascites. It is to be expected that the vital capacity would increase corresponding to the diminution of the amount of fluid in the pleural or peritoneal cavities. The findings in the patients we have studied in whom either hydrothorax or ascites, alone or combined, were present are given in Table 3.

In six of the eight cases outlined in Table 3 the hydrothorax and ascites disappeared while the patients were under observation. After the disappearance of the pleural and peritoneal fluid, the vital capacity continued to increase to the extent of from 4 to 6 per cent. in three cases, and 10, 12 and 36 per cent. in the other three. The findings indicate that the decrease in the vital capacity in these cases depended in part on the factors other than the hydrothorax and ascites.

Mackenzie has long believed that anginoid pain or cutaneous and muscular hyperesthesia were evidence of myocardial insufficiency. It is interesting to note that with the onset of such symptoms in two otherwise uncomplicated cases of mitral stenosis, the vital capacity dropped markedly, to rise again with the cessation of symptoms. In one of these cases there

was a rather sharp anginoid pain over the precordium. In the other case there was marked cutaneous hyperesthesia over the front of the left chest, over the pectoral muscles, and also over the trapezius muscle in the neck. Deep inspiration did not cause pain in either case. The vital capacity findings are given in Table 4.

Chart 1—Vital capacity in Case 11.

The vital capacity in a patient with paroxysmal tachycardia was studied during and after an attack. There were no evidences of organic heart disease, and during the paroxysm there were no symptoms referable to the heart. Sixteen hours after the onset of a paroxysm in which the heart rate was 180 per minute, the vital capacity was 58 per cent. Two days later, and twenty-four hours after cessation of the tachycardia, the vital capacity had increased to 83 per cent. A continued rapid rate of the heart in paroxysmal tachycardia is frequently accompanied by marked signs of myocardial insufficiency, but in our case the lowered vital capacity was the only indication of this. The accompanying charts show graphically the changes in the vital capacity in three typical cases (11, 12 and 19). Such charts are excellent objective records of the clinical course which each of the patients has passed through.

REPORT OF THREE TYPICAL CASES

CASE 11 (Med. No. 6261).—Chronic myocarditis, auricular fibrillation, hyperthyroidism.—Woman, aged 39, white, admitted, March 9, 1917, discharged, March 30, 1917, had had exophthalmic goiter for the last eight years. Six years ago she had a break in cardiac compensation, since which time her feet had been edematous at different periods. On entrance to the Peter Bent Brigham Hospital there were orthopnea, double hydrothorax and considerable edema of the legs. The usual findings of exophthalmic goiter were present. The area of cardiac dullness measured 19 cm. to the left in the fifth interspace and 2 cm. to the right in the fourth interspace. The cardiac action was absolutely irregular. A systolic murmur was audible over the region of the apex of the heart. Blood pressure was 142 mm. systolic and 90 mm. diastolic. The electrocardiographic diagnosis was auricular fibrillation.

Chart 2.—Vital Capacity in Case 12.

March 10, there was less edema of the legs than was present on admission.

March 12, the edema of the legs had subsided. There was less fluid in the pleural cavities.

March 16, hydrothorax was no longer present.

March 19, 22 and 26, the vital capacity had increased 10 per cent. since March 16. During that time the patient's physical condition had much improved. The pulse rate had

diminished from 80 to 90 to from 60 to 70 per minute, and the heart was well compensated.

CASE 12 (Med. No. 6152).—Mitral stenosis and insufficiency, myocardial insufficiency, auricular fibrillation, bronchitis, syphilis.—Man, aged 53, white, admitted, Feb. 15, 1917, died, Feb. 24, 1917, had been told ten years before that he had a valvular heart disease. During the last four years the patient had had occasionally a little dull precordial pain, and also some dyspnea on unusual exertion, and momentary attacks of vertigo. For several years his memory had been faulty, and he had become definitely irritable in disposition. The present illness began three months before with the symptoms of acute bronchitis and dyspnea, which had grown progressively worse. For the last week before admission to the Peter Bent Brigham Hospital the legs had been swollen. Physical examination showed marked orthopnea, anasarca, and deep cyanosis of the face, lips, ears and fingers. The area of cardiac dullness measured 14 cm. to the left in the fifth interspace and 45 cm. to the right in the fourth interspace. The heart's action was absolutely irregular, and there was a pulse deficit of 38 to the minute. There was a systolic murmur audible over the entire precordium, loudest in the region of the apex of the heart. In the longer pauses between heart beats a short early diastolic murmur was heard at the apex. Blood pressure was 130 mm. systolic and 90 mm. diastolic.

February 17, the patient was slightly worse. There was an increase in the edema of the scrotum, and the patient's cough

Chart 3—Vital capacity in Case 19.

was more frequent. However, there was less cyanosis than on admission.

February 19, the patient's condition was unchanged.

February 23, the patient was decidedly worse. He appeared to be much weaker physically. The cough was more frequent. He died several hours after the last vital capacity record had been obtained.

CASE 19 (Med. No. 6130).—Chronic myocarditis, hypertension, auricular fibrillation, chronic nephritis.—Man, aged 63, white, admitted, Feb. 12, 1917, discharged, March 13, 1917, had unimportant past medical history. The present illness began ten weeks before admission to the hospital with some generalized weakness, slight dyspnea, and fatigue on exertion. For the last three weeks the patient has been dyspneic. Physical examination revealed that the patient was orthopneic. There was considerable edema of the legs. There were coarse râles throughout both lungs. The area of cardiac dullness measured 16 cm. to the right in the fourth interspace. The heart's action was irregular, and the cardiac sounds were but faintly audible. No murmurs were heard. The aortic second sound was accentuated. Blood pressure was 190 mm. systolic and 90 mm. diastolic. An electrocardiogram showed the condition of auricular fibrillation to be present.

February 13, the patient was orthopneic. There was considerable edema of the feet and legs. Numerous coarse moist râles were present throughout both lungs. There was some cough.

February 14, there was much less edema of the feet and legs. Cough persisted.

February 16, the edema of the feet and legs had almost dis-

appeared. But few râles were present in the lungs. Cough persisted.

February 19, the patient lay comfortably in the prone position. The cough persisted.

No change occurred in the patient's condition until March 1. The cough was much less and there was little sputum.

March 3, 5, 8 and 12, the patient's condition remained stationary.

March 13, the patient was discharged from the hospital able to return to his work as a cabinet maker.

SUMMARY

Attention is called to the importance of a decrease in the vital capacity of the lungs as a factor in the production of dyspnea in heart disease. The degree to which the vital capacity is decreased below certain normal standards corresponds closely to the tendency to dyspnea. Since the tendency to dyspnea depends largely on the functional capacity of the heart, the determination of the vital capacity of the lungs may serve as an indirect measure of the cardiac condition. In a series of twenty-four cases it has been found that the clinical condition of cardiac patients varies closely with the changes in the vital capacity of the lungs. An improvement in the functional state of the heart is associated with a rise in the vital capacity. When the condition of the heart is apparently remaining stationary, changes in the vital capacity are not marked, and when there is evidence of increasing cardiac insufficiency, the vital capacity of the lungs falls. Charts showing the variations in the vital capacity of the lungs of patients with heart disease are frequently satisfactory objective records of the clinical course of the disease, and they may be of distinct aid in prognosis.

ABSTRACT OF DISCUSSION

DR. JOSEPH H. PRATT, Boston: Stimulated by Dr. Peabody's work, Dr. Paul D. White and I began a study of the vital capacity in patients at the cardiac clinic at the Massachusetts General Hospital. We have the records now of fifty patients. We find that the vital capacity corresponds closely to the functional condition of the heart as determined by the ordinary methods. Patients who were short of breath after walking slowly on a level for a distance of one or two blocks had usually a vital capacity of 50 per cent. or less. Ambulatory patients rarely have less than 40 per cent. In one case, however, it was 30 per cent. In diagnosis, the test is of great value in distinguishing between true cardiac weakness and those cases of cardiac neurosis that simulate organic disease of the heart. The difficulty in distinguishing between these two conditions is due to the fact that the first symptoms of cardiac weakness are usually subjective. The patient first notices slight breathlessness on exertion, and this may be present in organic heart disease before anything abnormal is found in the physical examination.

The value of the test was shown strikingly in the following case: A man had been treated in the dispensary for a year for myocardial disease. He complained that he was short of breath when he walked on the level a distance of 100 yards. Although nothing abnormal was found on the examination of the heart, the diagnosis had never been disputed. We found that his vital capacity was normal. From careful inquiry I learned that although he claimed to have a feeling of breathlessness while walking, he was able to run up stairs without any discomfort. This showed conclusively that the subjective sensation of which he complained was not due to cardiac weakness. We had mistaken a cardiac neurosis for organic heart disease.

The method is of importance in determining the functional capacity of the heart in patients who are in bed. Recently I had under observation at one time three patients, with mitral stenosis. All gave a history of shortness of breath on slight

exertion. In bed they exhibited no signs of cardiac insufficiency. They were able to lie with their heads low; there was no edema, and the pulse in each case was slow. To have tested their cardiac capacity by allowing them to exercise would have been too hazardous. The vital capacity test was made. Two had 40 per cent., the other about 80 per cent. I had previously suspected that there was a large neurotic element in the patient with the high vital capacity. Encouraged by the result of this test she has been taking recently, without discomfort, more exercise than she had for several years. The results of treatment among our ambulatory patients has not been encouraging as measured by the vital capacity test, and in only one case has there been a marked increase in the vital capacity from the use of digitalis, with the patient up and about. In this case, which was one of auricular fibrillation, the increase amounted to 25 per cent.

DR. T. B. BARRINGER, JR., New York: I am able in many ways to confirm the results which Dr. McClure has obtained. In estimating the vital capacity in cardiac patients I have found that it is much lower than in normal persons, and the greater the insufficiency, the lower the vital capacity. In cardiac patients with normal reserve power I have found that the vital capacity is normal. In cardiacs with low reserve power, but no insufficiency, the vital capacity is frequently normal. I have felt that the decrease in vital capacity in cardiac insufficiency was due to the congestion of the lungs which was present. In the treatment of patients by graduated exercises I have found that as the heart's reserve power increases and the clinical condition of the patient improves, the vital capacity goes up. Two years ago I drew attention to a small series of cases in which the average increase of vital capacity during the course of treatment was 25 per cent.

DR. R. G. PEARCE, Cleveland: I have an interesting case which Dr. Hoover and I saw in Lakeside Hospital, in which we estimated the vital capacity of the lungs and the cardiac output by the method proposed by Christianson, Douglas and Haldane, which determines the blood flow through the lungs per minute; that is, cardiac output of the right ventricle. The man had a badly decompensated heart. He had a vital capacity of about 1.2 liters and a cardiac output of 3 liters. After a course of digitalis for three days he had a vital capacity of 1.8 liters and a blood flow per minute of 4 liters. After a week he had 2.4 liters vital capacity and a blood flow of 4.5 liters per minute. The normal man has a cardiac output of 5 or 6 liters per minute. I think this observation is interesting because it checks up cardiac output with vital capacity.

DR. C. W. McCLURE, Boston: A study has been made of cardiac patients who were compensated, and it has been found, as I tried to bring out early in the paper, that the vital capacity in patients with cardiac disease who we say are compensated varies in different patients. If the patient can lead a normal life, the vital capacity is almost invariably above 50 per cent. If the patient is able to do a slight amount of work, but must lead a restricted life, the vital capacity will fall almost invariably to somewhere between 70 per cent. and 90 per cent. of the normal. Patients whose vital capacity is less than 70 per cent. are dyspneic on very moderate exertion. The lower the vital capacity, the easier the patient becomes dyspneic, and patients who are down near 40 per cent. are usually decompensated or become decompensated very easily.

A New Method of Cleaning Slides.—The method of cleaning old used slides given below is new so far as the writer knows, and as it is simple, cheap and effective, it seems to merit publication. The only reagent necessary is commercial (household) ammonia used full strength. The slides are soaked in this indefinitely (at least twenty-four hours), when it will be found that they can be rinsed in water and wiped clean. Stained smears of all kinds covered with immersion oil and tissue sections mounted in balsam are all equally well cleaned. The immersion oil need not be removed before soaking, although it is advisable to wipe off the excess. The ammonia may be used repeatedly if kept in a receptacle with a tight cover to prevent loss.—REUBEN A. JOHNSON, M.D., Minneapolis.